CONTRIBUTORS.

CLARK, C. F., M.D.

COLLINS, E. TREACHER, F.R.C.S. Eng.

DUANE, ALEXANDER, M.D.

ELLETT, EDWARD C., M.D.

GIFFORD, H., M.D.

POSEY, WILLIAM CAMPBELL, M.D.

REEVE, R. A., M.D.

SHUMWAY, EDWARD A., B.S., M.D.

SPICER, T. HOLMES, F.R.C.S. Eng.

STARR, ELMER G., M.D.

SUTER, WILLIAM NORWOOD, M.D.

VEASEY, CLARENCE A., A.M., M.D.

WEEKS, JOHN E., M.D.

WOOD, CASEY A., M.D.

WÜRDEMANN, H. V., M.D.

A TREATISE

ON

DISEASES OF THE EYE.

FOR STUDENTS AND PRACTITIONERS.

BY VARIOUS AUTHORS.

EDITED BY

WILLIAM CAMPBELL POSEY, A.B., M.D.,

PROFESSOR OF OPHTHALMOLOGY IN THE PHILADELPHIA POLYCLINIC; SURGEON TO THE WILLS EYE HOSPITAL; OPHTHALMIC SURGEON TO THE HOWARD AND EPILEPTIC HOSPITALS; MEMBER OF THE AMERICAN OPHTHALMOLOGICAL SOCIETY.

VOLUME I.

OF

A TREATISE ON DISEASES OF THE EYE, NOSE, THROAT, AND EAR.

EDITED BY

WILLIAM CAMPBELL POSEY, M.D., AND JONATHAN WRIGHT, M.D.

ILLUSTRATED WITH 358 ENGRAVINGS AND 19 PLATES IN COLORS AND MONOCHROME.

PROSUNT QUAE OMNIBUS

LEA BROTHERS & CO.,
PHILADELPHIA AND NEW YORK.

DORNAN, PRINTER.

PREFACE.

THE close anatomical and pathological relationship existing between the Eye, Nose, Throat and Ear, and the fact that many American physicians combine these specialties in their practice, have led to the preparation of this work. To suit the convenience of all readers it is issued either in one or in two volumes. In the two-volume edition the first considers the Eye, and the second the Nose, Throat and Ear. The volumes are procurable separately.

The Editors believe that the uniform plan adopted for the work as a whole is calculated to produce a homogeneous and effective result. The distribution of subjects has been arranged along logical lines of division, so that each author was enabled to treat his department in its entirety, a distinct advantage in the way of avoiding repetition and confusion. This simplification has resulted in the additional benefit of a moderate number of chapters. Their authorship has been confided to contributors each of whom had previously demonstrated his special ability in connection with the subject assigned. Separate chapters on Anatomy and Physiology have been omitted, as such general knowledge is presupposed, but enough information will be found in connection with each subject to explain the pathology and symptomatology.

It has been the aim of the Editor of the volume on the Eye to present to the profession a work which might be not only a practical text-book for students, but also, by reason of its containing the most recent utterances of eminent investigators in ophthalmic science, a reference of value for eye specialists.

By an arrangement of the subject whereby the reader is brought into immediate clinical relationship with the patient, by the avoidance of unnecessary optical formulæ and principles, and by the introduction of a

comprehensive chapter upon the Eye in Relation to General Diseases, an effort has been made to adapt the work to the needs of general practitioners as well, and it is hoped that these members of the profession may be enabled to acquire from its pages a knowledge of the eye which is so indispensable to the proper recognition and treatment of many forms of systemic disease.

LIST OF CONTRIBUTORS.

CLARK, C. F., M.D.,

Professor of Ophthalmology and Otology in the Starling Medical College; Attending Eye and Ear Surgeon to the St. Francis Hospital, and Consulting Surgeon to the Children's Hospital, Columbus, Ohio.

COLLINS, E. TREACHER, F.R.C.S. Eng.,

Surgeon to the Royal London Ophthalmic Hospital (Moorfields); Ophthalmic Surgeon to the Charing Cross Hospital, and Lecturer on Ophthalmology at the Medical School.

DUANE, ALEXANDER, M.D.,

Instructor in Ophthalmology and Surgeon to the Dispensary of the Cornell Medical College, New York; Surgeon to the Ophthalmic and Aural Institute; Ophthalmologist to the Samaritan Home; Visiting Ophthalmologist to the Randall's Island Hospital, New York.

ELLETT, EDWARD C., M.D.,

Ophthalmic and Aural Surgeon to the St. Joseph's Hospital, the City Hospital, and the Leath Orphan Asylum, Memphis, Tenn.

GIFFORD, H., M.D.,

Professor of Ophthalmology and Otology in the University of Nebraska, Omaha, Nebraska.

POSEY, WILLIAM CAMPBELL, M.D.,

Professor of Ophthalmology in the Philadelphia Polyclinic; Surgeon to the Wills Eye Hospital; Ophthalmic Surgeon to the Howard and Epileptic Hospitals, Philadelphia; Member of the American Ophthalmological Society.

REEVE, R. A., M.D.,

Professor of Ophthalmology and Otology in the University of Toronto; Ophthalmic and Aural Surgeon to the General Hospital and Hospital for Sick Children, Toronto, Canada.

SHUMWAY, EDWARD A., B.S., M.D.,

Ophthalmologist to the Presbyterian Home for Widows and Single Women of Pennsylvania; Voluntary Associate in the William Pepper Laboratory of Clinical Medicine, Philadelphia.

SPICER, T. HOLMES, F.R.C.S. Eng.,

Surgeon to the Moorfields Eye Hospital; Ophthalmic Surgeon to the St. Bartholomew's Hospital, London, England.

STARR, ELMER G., M.D.,

Clinical Professor of Ophthalmology in the Medical Department of the University of Buffalo; Surgeon to the Eye and Ear Department, University Dispensary, Buffalo, N. Y.; Eye and Ear Surgeon to the Erie County Hospital, German Deaconess' Hospital, and Buffalo Hospital of Sisters of Charity, Buffalo, N. Y.

SUTER, WILLIAM NORWOOD, M.D.,

Assistant Surgeon to the Episcopal Eye, Ear, and Throat Hospital, Washington, D. C.

VEASEY, CLARENCE A., A.M., M.D.,

Demonstrator of Diseases of the Eye in the Jefferson Medical College; Assistant Ophthalmic Surgeon in the Jefferson Medical College Hospital; Ophthalmic Surgeon in the Methodist Episcopal Hospital; Consulting Ophthalmologist in the Philadelphia Lying-in Charity.

WEEKS, JOHN E., M.D.,

Surgeon to the Ophthalmic Department of the New York Eye and Ear Infirmary; Professor of Clinical Ophthalmology in the University and Bellevue Hospital Medical College; Fellow of the New York Academy of Medicine; Member of the American Medical Association.

WOOD, CASEY A., M.D.,

Professor of Ophthalmology in the Chicago Post-graduate Medical School; Professor of Clinical Ophthalmology in the University of Illinois; Ophthalmic Surgeon to the Passavant Memorial Hospital and to St. Luke's Hospital, Chicago; Consulting Ophthalmologist to St. Anthony's Hospital, Chicago, Ill.

WÜRDEMANN, H. V., M.D.,

Professor of Ophthalmology to the Milwaukee Medical College, and Chief of Eye Clinic; Ophthalmic and Aural Surgeon to the Trinity Hospital, to the Milwaukee Children's Hospital, to the Milwaukee County Hospital, and to the Milwaukee County Hospital for the Chronic Insane.

CONTENTS.

THE EYE.

CHAPTER I.

CHAPTER II.

CHAPTER III.

CHAPTER IV.

CHAPTER V.

CHAPTER VI.

CHAPTER VII.

CHAPTER VIII.

CHAPTER IX.

CHAPTER X.

CHAPTER XI.

CHAPTER XII.

CHAPTER XIII.

CHAPTER XIV.

CHAPTER XV.

THE EYE.

THE EYE.

CHAPTER I.

EXAMINATION OF THE EYE.

By WILLIAM CAMPBELL POSEY, M.D.

General Considerations. The student of medicine should approach the study of the eye with a twofold purpose: first, to obtain through it further information regarding the state of the general system, and, secondly, to become acquainted with the morbid processes which attack one of the most important organs of the body. There is no other organ in the body in which the general systemic condition can be studied to better advantage than the eye, for it presents in a compact form representation of nearly all the tissues of the body, and by reason of the transparency of some of its coats the student is enabled actually to witness physiological and pathological processes occurring within it. A living nerve head, the optic papilla, and the retinal vessels are unfolded to the gaze of the ophthalmologist, and an opportunity afforded him of observing the perfect cycle of the supply of an organ with arterial, and the escape of its venous, blood.

For the proper study of this important organ it is essential that the student proceed systematically and thoroughly, for while it often happens that a trained clinician is enabled by the brief recital of symptoms or by a rapid glance at the eye to make a proper diagnosis, it is better that the student, who has yet to attain experience and skill, should follow some settled order of investigation, and that for the purpose of future reference, as well as to ensure accuracy, he should accustom himself to record faithfully all his observations in an appropriate case-book.

Inspection of the General Physical Condition. Before proceeding to an immediate inspection of the eye itself, it is advantageous that the general physical condition of the patient should be taken account of. For this purpose he should be seated in a chair facing a window, the student, with his back to the light, seating himself several feet distant from the patient. Under this strong illumination the entire person of the patient should be rapidly inspected, and any departure from the normal in the exposed portions of the skin and of the glandu-

lar system, as well as the character of any eruption, noted. Indications of anæmia or plethora should be searched for, and any evidences of jaundice. Finally, the general expression of the patient and the presence or absence of pain, or of any intolerance to light, should be taken into account.

Inspection of the Eyes and Their Adnexæ. This general survey of the case being completed, the attention of the student should be directed more especially toward the region of the eyes. The shape and general configuration of the head and the character of the wrinkles in the skin of the forehead and at the root of the nose should be studied. Any tendency toward facial asymmetry should be noted, and comparison made whether both orbits are on the same horizontal plane, and whether their cavities are deep or shallow. The degree of prominence and the size of the eyeballs should be remarked, and the relationship which the eyes bear to one another. Especially should the presence or absence of inflammation of the eyeballs be taken into account; if but one eye is diseased, its condition should be compared with that of the sound eye, as comparative examinations of this kind are frequently of great value. The student should carefully scan the region of the sinuses accessory to the eye, to detect swelling or signs of inflammation in them. Any signs of previous injury about the eyes should be recorded. The attention should then be directed particularly to the lids as to any inversion or eversion of their edges, or thickening or distortion or swelling of them; estimation should be made of abnormal narrowness or width of the palpebral fissures. The action of the orbicularis in closing the lids should be tested, and any twitching of the lids and associated muscles of the face noted. The region of the inner canthus should be inspected for evidences of swelling, or retained tears, or other signs of faulty drainage in the lacrymal apparatus.

Having observed the general appearance of the patient, and having obtained by the inspection of the region of the eyes in a general way some idea of the nature of the ocular complaint, before attempting a closer inspection of the eye the student should next obtain by careful questioning a precise and complete history of the patient's family and personal history.

Family and Personal History. The sex, race, and age of the patient should be recorded, and account made of the nature of the occupation, certain callings, by reason of the accidents to which they expose the eyes, and others, by the enforced strain which they place upon them, being particularly liable to produce ocular lesions. Inquiry should also be made into the marital relations; and if the patient be married, of the number and health of any offspring. Any hereditary tendency, particularly to ocular disease in the ancestry, should be recorded, also the temperament of the patient, whether it be sanguine or the contrary, in order to ascertain the value and degree of reliance to place upon the patient's statements regarding the severity of the symptoms, nervous subjects exaggerating and lymphatic ones sup-

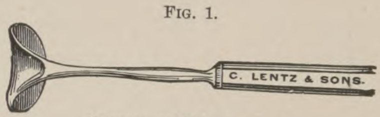
pressing their sensations. The habits should be inquired into, especially regarding the use of alcohol and tobacco. If syphilis be suspected, question should be made regarding the primary sore, as well as the time of appearance of any secondary manifestations. All previous illnesses should be taken account of, especially of the existence of gout, rheumatism, tuberculosis, malaria or other dyscrasia. The urine should be tested in all doubtful cases, and its examination made a part of the routine in all cases requiring important operations upon the eyeball, such as cataract. If the patient be a female, she should be questioned regarding menstrual disorders, and particularly as to the influence of the menstrual epoch upon the ocular symptoms. Finally, should the inspection of the patient have aroused suspicion as to involvement of the central nervous system, inquiry should be made of all possible sensory and motor disturbances.

Ocular History. Having obtained by direct and searching cross-questioning a precise knowledge of the antecedents as well as of the personal history of the patient, the student is now prepared to direct his inquiries to the ocular condition itself. He will accordingly inquire as to the time and manner of onset of the present attack; whether it was accompanied by pain or inflammatory symptoms, the degree to which vision was disturbed, and whether one eye or both were affected. He will inquire into previous attacks of ocular inflammation, and trace any relationship with the present outbreak. Should the case be one of refraction error, the previous wearing of glasses and their efficacy in relieving the ocular symptoms should be recorded, also regarding the location and character of any head pain and the influence of the use of the eyes in reading upon it. If it be apparent that a palsy of one or more of the extra-ocular muscles be present, the nature of the double vision should be elicited. In fine, the student cannot be too searching nor too persistent in his questioning, and should exhaust every possible phase of the subject before approaching the direct and closer inspection of the eye and its appendages.

Direct Inspection of the Eye and Its Appendages. For this purpose it is necessary that the student should approach the patient sufficiently close to observe the finer structures of the eye and to permit of any manipulation, either with the hand or with instruments, that may be required; he should, moreover, refrain from handling the eye any more than is necessary, and endeavor to gain as much information as is possible by inspection with the naked eye, without the intervention of lenses or instruments, for such aids are not always at hand, and, moreover, even the lightest touch is often sufficient to render sensitive eyes so irritable that further examination is impossible. In many young children, and in individuals who have an intense intolerance to light, however, inspection without handling is fruitless, on account of the tightly closed lids, so that the observer will be compelled to open them himself before he can obtain a view of the eye. This is best accomplished

in young children by the operator seating himself in such a manner that the light from a window falls upon his right or left side, while the child's head is held firmly between his knees, the body being supported upon the lap of an attendant, who should also grasp the hands, the legs being left free. The head being thus rendered immobile, the surgeon can inspect the neighboring parts deliberately, and can examine the eye satisfactorily by drawing the lids slowly apart, by pressing on the inferior and superior orbital ridges, or by inserting a Desmarres lid elevator (Fig. 1) beneath them, always exer-

FIG. 1.



Desmarres' lid retractor.

cising the greatest care to avoid pressure upon the eyeball itself, for fear of injuring the cornea. When there is marked intolerance to light, a 4 per cent. solution of muriate of cocaine may often be successfully employed to allay irritation, although in some cases general anæsthetization by chloroform may have to be resorted to before a satisfactory examination can be made. In adults it is possible to examine even the most sensitive eyes by making gentle traction on the lids, by drawing them toward the inferior and superior orbital ridges, thereby avoiding pressure upon the eyeball itself.

The Lids. The character of any changes which have been noted in the lids during the general inspection should now be studied more carefully, especial care being devoted to the condition of their margins, as to misplaced cilia or the presence of pediculæ, and the character of any incrustation or swellings.

Lacrymal Apparatus. The region of the inner canthus should be inspected most rigorously, any localized injection of the conjunctiva or collection of tears or mucus at that point exciting the suspicion of obstruction in the proper canalization of the secretion from the eye. The position and patulency of the lacrymal puncta should be ascertained, and gentle pressure made with the tip of the finger over the lacrymal sac, with a view to expressing any retained contents.

The Orbit and the Position of the Eyeball in It. Unequal prominence of the globes may be measured by placing the straight edge of a card from the supra-orbital ridge to the cheek, and comparing the distance of the cornea from the card on the two sides. Palpation of the orbit should be practised by passing the index finger along the bony margins of the orbit, the finger being allowed to dwell particularly over the region of the lacrymal gland, to detect any enlargement or unevenness. Pressure over the foramina of exit of the supra-orbital and infra-orbital nerves should not be omitted.

The Conjunctiva and Its Cul-de-sacs. Before exploring the recesses of the cul-de-sacs, the caruncle and the semilunar fold in the angle of

the inner canthus should be examined for small growths or foreign bodies. The conjunctiva of the lids, *palpebral conjunctiva*, should then be inspected, and any change in its vascularity or in the character of its secretion, and the presence of granulations or foreign bodies, noted. To examine the conjunctiva of the *retrotarsal folds* and the cul-de-sacs, it is necessary to evert the lids; this is readily accomplished in the case of the lower cul-de-sac by drawing the lower lid gently down with the index finger of the right hand, while the patient is told to direct his gaze upward. Inspection of the upper cul-de-sac is less simple, and is performed by grasping the edge of the upper lid and a few cilia with the thumb and index finger of the right hand, and by depressing the upper edge of the cartilage of the lid with a finger of the left hand, or with some convenient instrument, such as a probe, while the patient looks steadily downward. By requesting the patient to direct his gaze still further downward the palpebral portion of the lacrymal gland may be brought into view. The *bulbar conjunctiva* is ordinarily invisible save for the few bloodvessels which are distributed through it. The color of the sublying sclera should be noted, and any undue vascularity and prominences taken account of.

Before proceeding further, it is desirable that the student should have a clear idea of the vascular supply of the exterior of the eye, in order that he may appreciate the different forms of congestion peculiar to the vessels of the several tissues, as no other symptom gives surer indication of the location of ocular lesions.

Bloodvessels of the Exterior of the Eye. The vascular supply of the exterior of the eye may be grouped for convenience into three systems: 1. *The Posterior Conjunctival Vessels*, or the vessels proper to the conjunctiva. 2. *The Anterior Ciliary Vessels*. These consist of (*a*) perforating arteries and veins, and (*b*) non-perforating arteries and veins. The perforating arteries supply the sclerotic, iris, and ciliary body, their veins receiving the blood from the canal of Schlemm and the ciliary body. These vessels are visible in health as several comparatively large tortuous vessels which perforate the globe about 5 mm. from the corneal limbus. The non-perforating or episcleral vessels, which are branches from the anterior ciliary vessels, are very numerous and form a zone of closely set vessels around the cornea. They are invisible in health. 3. *The Anterior Conjunctival Vessels and their Loop-plexus on the Corneal Border*. These are the vessels proper to the margin of the cornea and immediately adjacent zone of conjunctiva, and it is by means of these numerous minute branches which are offshoots of the anterior ciliary vessels that systems 1 and 2 anastomose. (Plate I., Fig. 1.)

Conjunctival congestion is the name given to indicate that form of congestion which is caused by an injection of the posterior conjunctival vessels. The injection is most marked at the fornix and its immediate neighborhood, where these vessels are most numerous, and is less noticeable around the cornea. The vessels being situated

in the conjunctiva, may be made to slide readily over the globe, and are easily emptied by pressing on the lid with the finger. In this form of congestion the conjunctiva assumes a yellow or brick-red hue, especially in the region of the inner canthus, and there is more or less mucopurulent discharge. Conjunctival congestion is diagnostic of conjunctivitis. (Plate I., Fig. 2.)

Ciliary or circumcorneal congestion is the name given to an injection which is most marked in the zone immediately around the cornea, and gradually fades at the periphery of the globe. It is caused by injection of the anterior ciliary and anterior conjunctival vessels. As these are situated beneath the conjunctiva, they cannot be displaced or made to disappear by pressure on the lid. There is no accompanying discharge. In this form of congestion the circumcorneal region assumes either a pinkish or a scarlet hue, while in other cases deep-seated patches of a lilac or violaceous color appear. Ciliary injection indicates disease in the cornea, iris, or ciliary body; when the peculiar lilac-colored patches are present, disease of the deeper lying tissues, the sclera, and ciliary body is indicated; and when observed in connection with enlargement of the episcleral veins, a chronic increase of intra-ocular tension—glaucoma—may be suspected. (Plate I., Fig. 3.)

Mixed Forms of Congestion. On account of the free anastomosis of the three groups of vessels, it frequently happens that there is merging of the different types of congestion into one another. It should be borne in mind, however, that while a prolonged ciliary congestion gradually produces more or less conjunctival congestion, the converse is not true, for a conjunctivitis will not excite a ciliary injection unless the cornea or iris also is affected.

The Cornea. The shape, the general curvature of the cornea, and the position, extent, and density of all irregularities and opacities in it, should be studied on account of the important bearing which they have upon the vision and refraction of the eye. For this purpose two methods are available: examination of the corneal reflex and direct inspection.

Examination of the Corneal Reflex. When the light from a window is permitted to fall directly upon the cornea, and the eyes are made to follow the finger of the surgeon while it is moved in various directions, it will be noticed, if there be any irregularity upon the surface of the cornea, that the image of the window bars which is thrown upon it, instead of being reflected clear and well defined, will be broken and ill defined at these points. The same principle is made use of in the application of the Placido disk. (Fig. 2.) This consists of a target on which are concentric alternate black and white circles, with a central perforation. In its employment, the patient should be placed with his back to the light, the surgeon viewing the reflection of the image of the circles upon the cornea through the opening in the centre of the disk. Any irregularity or excessive difference in the curvature

of the meridians of the cornea will be manifested by a break or distortion in the circles. The *corneal reflex* should always be studied

FIG. 2.

Placido's disk, or keratoscope.

as part of the routine ophthalmoscopic examination, the observer stationing himself for this purpose behind and to one side of the patient's head, so as to have the patient's face in shadow. The light

FIG. 3.

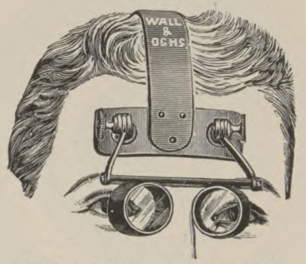
Corneal loupe.

is then thrown upon the eye by a plane mirror, and the shadows carefully studied through a hole in the mirror. (*Vide* Retinoscopy.)

The most accurate and exhaustive method of studying the shape of the cornea by utilizing the reflex that it casts is by means of the ophthalmometer. (*Vide* page 114.)

Examination by Direct Inspection. This may be done with the naked eye, by means of a monocular magnifying lens (the corneal loupe)

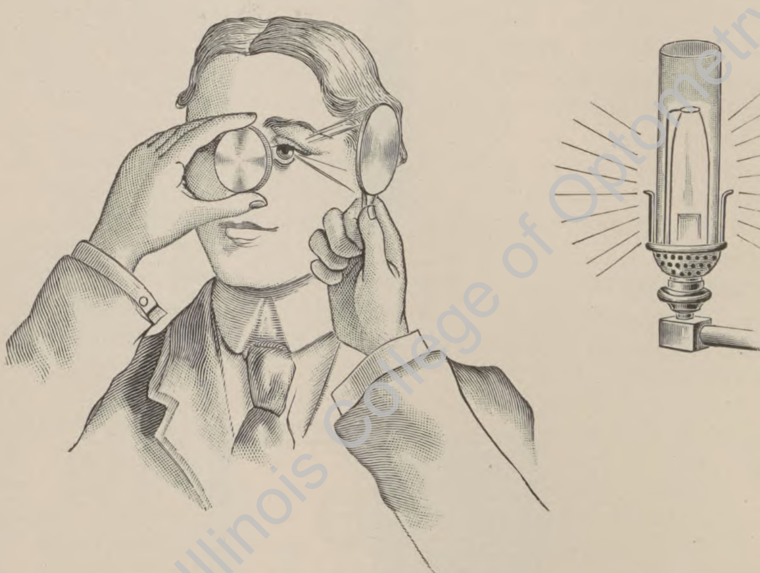
FIG. 4.



Jackson's binocular magnifier.

(Fig. 3), by a binocular magnifier (Jackson's) (Fig. 4), by oblique illumination, and, finally, if it be the purpose to study minute changes in the cornea, a compound microscope may be used which has been specially constructed for this purpose.

FIG. 5.



Oblique or focal illumination.

In the examination of the cornea by *oblique* or *focal illumination*, two convex lenses of several inches focus are necessary, one to be used to concentrate the light upon the cornea, while the other is

employed as a magnifier, through which the illuminated surface may be studied. (Fig. 5.) The patient should be seated in a dark room with the light on the temporal side and slightly in front of the plane of the patient's face. This method is extremely valuable, for by varying the distance of the lens from the eye it is possible to study not only changes in the cornea, but also those in the anterior chamber, iris, and lens; and if the pupil be dilated and the light thrown almost perpendicularly into the eye, changes in the anterior layers of the vitreous may be made out as well.

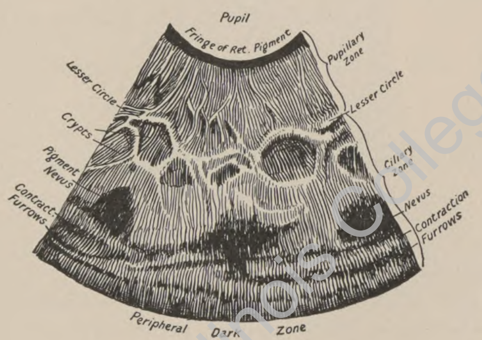
Loss of substance in the corneal epithelium may be demonstrated by instilling a drop of fluorescin into the eye (Gruebler's fluorescin, 2 per cent.; carbonate of sodium, 3.5 per cent.), the surface from which the epithelium is removed being stained greenish yellow by the drug, while the rest of the membrane remains clear.

The sensitiveness of the cornea is tested best by gently touching it with a wisp of cotton: if sensation be unimpaired, the eye will wink reflexly; but if the lids remain immobile, further investigation of the sensibility of the skin of the surrounding tissue should be made with an æsthesiometer, to determine the extent of the anæsthesia.

The anterior chamber should be examined in respect to its depth and contents; among the latter which the chamber may contain being blood, or *hyphæma;* pus, or *hypopyon,* and foreign bodies.

The Iris. In the examination of the iris, the attention should be directed chiefly to its color, to the appearance of its stroma, and especially to the size, position, and behavior of the pupil. The color of the iris is due to the amount and distribution of the pigment in it; in albinism, where there is an absence of pigment, the iris is translucent,

FIG. 6.



The Iris. (FUCHS.)

and in newly born children it is almost invariably of a light grayish blue. The irides may differ in color in the two eyes, *chromatic asymmetry;* or parts of the same iris may be colored differently, *piebald iris.* Discoloration of the iris should always excite suspicion of inflammation of membrane.

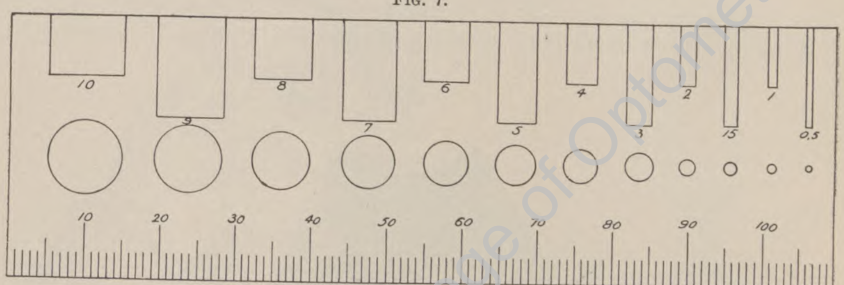
When viewed through a magnifying glass, with the aid of oblique illumination, the iris is seen to be composed of a series of elevations (Fig. 6) and depressions, the former being occasioned by the blood-vessels, which run radially from the base of the iris to the pupil; while the depressions correspond to crypts in the stroma of the iris, and are found chiefly near the pupillary margin. Although these elevations and depressions are sharp and distinct in the normal eye, they become blended in inflammation, which constitutes an important sign of iritis.

Changes in the plane of the iris, tears in its pupillary edge and base, and any wave-like movements on its surface, *iridodonesis*, should be searched for carefully. Thickening and vascularity of the membrane should be remarked and the character of any nodulation noted.

The Pupil. The chief characteristics of the healthy pupil are its circular outline and its mobility.

The size of the pupil varies greatly in health, ranging from 2.44 to 5.82 millimetres, being influenced by age and refraction, and is directly dependent upon the stimulation of the light, accommodation and convergent impulses which it receives. Woinow places the average at 4.14 millimetres. As a rule, age causes the pupil to grow smaller, and it is also more likely to be smaller in hypermetropia than in myopia. Its width may be ascertained by means of the pupillometer, which consists of a scale, preferably of glass, graduated in circles ranging from 1 to 8 millimetres. (Fig. 7.) This is held close to the eye, and while

FIG. 7.



Hirschberg's pupillometer.

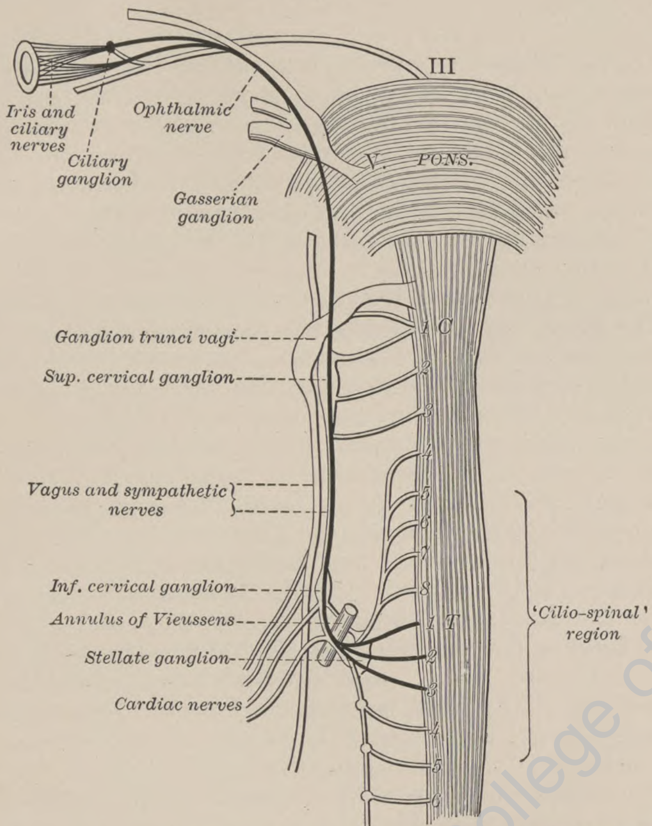
the patient fixes his gaze upon some distant object, the circle should be found which corresponds with the diameter of the pupil. Until the student has acquired sufficient skill to enable him to obtain an accurate measurement of the pupil by simple inspection, some such scale should be employed in all cases.

The pupil should be round, but this is usually prevented by astigmatism, which gives it an oval appearance; it should also be situated slightly to the nasal side of the centre of the cornea.

The separation of the pupils from each other varies with age and sex and with the form of the face; in adults it has an average of 58 mm., although Nagel places it at 63 mm.

The pupil is rarely clear black, the anterior surface of the lens reflecting some light; indeed, this reflex is often so marked in elderly subjects that the grayish film due to sclerosis of the lens is often mistaken for cataract by the inexperienced observer. If oblique illumination be employed, however, the true nature of the opacity in the lens becomes manifest.

FIG. 8.



Illustrating the paths of innervation of the iris. Constrictors from the corpora quadrigemina by the third nerve, ciliary ganglion and nerves to the circular muscles of the iris. Dilators from the bulb and cord by anterior roots of the first three thoracic nerves, especially the second, rami communicantes, cervical sympathetic and ganglia, Gasserian ganglion, ophthalmic branch of the fifth nerve, ciliary ganglion and nerves radiating to muscles of iris. (WALLER.)

The iris is the diaphragm of the eye, and by its action in intercepting marginal rays it prevents an excessive amount of light from entering the eye. In order that this may be accomplished with great rapidity and the size of the pupil instantly changed, the iris is provided with a delicate mechanism wonderfully adapted to the function which it has to perform. This consists of two antagonistic factors: one, a constricting mechanism, to contract the pupil; the other, a dilating one, to dilate

the pupil. The former is accomplished by stimulation of the oculomotor nerve and consequent contraction of the sphincter pupillæ, a circular muscle surrounding the pupil; the latter, by the absence of this stimulation and by the contracting effect of the cervical sympathetic, which supplies the dilator pupillæ, the meridianal muscular fibres of the iris and also the bloodvessels of the iris, contraction of these vessels causing narrowing of the iris and dilatation of the pupil.

The reaction of the pupil is either a *reflex* action, in which event the impulse passes along the optic nerve, the afferent nerve, to the oculomotor centre, and along the oculomotor nerve, the afferent nerve to the eye, through the medium of fibres which connect the corpora quadrigemina with the nuclei of the oculomotor nerve; or it may be *associated*, in which case the impulses are set into action simultaneously with efforts at accommodation and convergence. The degree of the pupillary contraction in associated action is always less than that observed in the reflex reaction.

THE REFLEX REACTION of the pupil may be either *direct* or *consensual*. *The direct light reflex* is the contraction of the pupil which is observed in the eye when it is exposed to increased illumination, the *consensual* or *indirect light reflex* being that which occurs in the pupil of the other eye, following exposure of one eye to light.

The direct light reflex is tested by alternately shading and uncovering the eye in daylight with the hand, or by concentrating artificial light upon it, either by means of oblique illumination or by the mirror of an ophthalmoscope, the gaze of the patient being fixed on a distant object, to avoid any associated stimulus from either accommodation or convergence stimuli. It sometimes happens that after the cover is removed from the eye the first contraction of the pupil to light is followed by dilatation, and often an interval of extreme contraction, being succeeded by moderate dilatations and contractions until the pupil becomes stationary. This condition is termed *hippus*, and is explained by Swanzy as follows: Each contraction of the pupil, by diminishing the supply of light to the retina, contains in itself the cause of the succeeding dilatation; and for the converse reason, each dilatation sets agoing the succeeding contraction, until at last equilibrium is attained. Hippus is seen in cerebro-spinal sclerosis, disseminated sclerosis, neurasthenia, hysteria, psychical disturbances, epilepsy, and acute meningitis in its early stages.

The consensual or indirect pupillary reaction is tested by observing the motions of the pupil in the other eye while the eye under examination is being alternately covered and uncovered. This test is dependent anatomically upon the fact that fibres pass from the retina of each eye through the chiasm partly into the right and partly into the left optic tract, and that from these the stimulus is transmitted directly to both right and left oculomotor nuclei, each nucleus setting up a contraction of the pupil of its own side.

The direct light reflex is tested for the purpose of detecting the existence of adhesions of the iris to the capsule of the lens (posterior synechia), and to determine the sensitiveness of the retina and of the visual apparatus generally to light. The reaction is an exceedingly delicate one, and indicates the presence or absence of quantitative perception to light. It will presently be explained, however, that the latter function may be wanting in certain diseased states, and yet the pupil reflex take place; or the pupil reflex may be wanting, and perception of light still be present.

The associated reaction of the pupil, or the accommodation and convergence reflex, is tested by requesting the patient to look fixedly at an object held in the median line about 10 cm. in front of the face. The contraction of the pupil which follows is due to the intimate association of the central innervation of the sphincter muscle of the iris, the ciliary body, and the internal rectus muscles. While accommodation unassociated with convergence will not cause contraction of the pupil, reaction follows convergence stimuli alone.

In contradistinction to the contraction of the pupil when acted upon by light or accommodation or convergence stimuli, *the pupil invariably dilates when acted upon by sensory stimuli.* Thus the pupil which is contracted during sleep and deep narcosis dilates at the moment of waking. The pupil dilates also under nervous excitement, such as fear and surprise, and also with deep inspirations and expirations; it is dilated also during hunger and in anæmia. Irritating or pinching the skin of the neck is followed also by pupillary dilatation (pain reaction). Both pupils should be equal in size, unequal pupils (*anisocoria*), although frequently of no import, being often a grave symptom. In testing the reflexes, it is essential to observe whether contraction and dilatation of the pupil occur simultaneously and to the same degree in both eyes. As a rule, it may be stated that the least movable pupil belongs to the affected eye.

As deeply seated disease of the brain and spinal cord frequently manifest themselves in some disturbance of the pupil, it is most essential for the student to understand fully its nervous connections.

The Behavior of the Pupil in Disease. Pathological processes which affect the iris manifest themselves either in a contraction of the pupil (*myosis*), or in a dilatation of it (*mydriasis*). Both of these differences in the diameter of the pupil may be the expression of either spasm or paralysis of the musculature of the iris, or they may be the result of some inflammatory condition of the iris or within the eyeball, as, for example, the myosis which accompanies iritis, or the mydriasis which is seen in glaucoma.

Myosis (contraction of the pupil). Myosis may be due either to spasm of the sphincter pupillæ or to an irritation of the contracting centre or nerve fibres, *spastic myosis;* or it may be the result of paralysis of the dilating fibres of the pupil or of the pupil-dilating centre or nerve fibres, *paralytic myosis.* Either cause operating alone occasions a moderate contraction of the pupil; if both are active, the

pupil is contracted to a pinpoint. Contraction of the pupil follows simultaneous stimulation of both dilating and contracting mechanisms. The most common cause of contracted pupil in disease of the eye is adherence of the iris to the lens capsule.

Spastic myosis is symptomatic of inflammatory affections of the brain and its meninges; it is present in the early stages of intracranial tumors which involve the third nerve; it is seen at the beginning of hysterical and epileptic seizures. Pressure upon the pons causes myosis. It results from stimulation of the pupillary contracting centre, and occurs in those who suffer from tobacco amblyopia and in those who follow trades which demand long maintained efforts of accommodation (watchmakers, jewelers, etc.). If, in the course of a case of cerebral disease, myosis gives way to sudden dilatation, the prognosis becomes grave, the stage of depression with paralysis of the third nerve being indicated. Myosis may be a reflex action in ciliary neurosis; it accompanies many diseased conditions of the portion of the eye supplied by the fifth nerve. The pupil in irritation myosis is but little affected by reflex stimuli; it is very susceptible, however, to drugs, mydriatics dilating it widely, and myotics contracting it *ad maximum.* In contradistinction to this, the pupil in paralytic myosis reacts actively to the different reflex stimuli, and is but little affected by mydriatics, although myotics contract it greatly.

Paralytic myosis occurs in spinal lesions above the dorsal vertebræ, and is especially significant of tabes dorsalis. In the early stages of this disease, in which the cilio-spinal centre or the higher region of the cord alone have been affected, the pupil is but moderately contracted, and reacts to both light and on convergence; later on, the pupil presents the phenomena which have been characterized as *Argyll-Robertson pupil* or *reflex iridoplegia—i. e.,* the pupil responds very slightly or not at all to light, but is active in accommodation and convergence. The lesion which produces the Argyll-Robertson pupil has been variously situated in the fibres which pass from the proximal end of the optic nerve to the oculomotor nuclei, and to a nuclear lesion pure and simple. Another pupillary sign which is seen in tabes dorsalis is known as *unilateral reflex iridoplegia.* In this condition one pupil reacts to accommodation, but not to light, while the pupil in the fellow eye responds normally. It is probably the result of a lesion in the nucleus of the sphincter of the iris.

Paralytic myosis is seen in general paralysis of the insane, in myelitis of the cervical portion of the cord, in paralysis of the cervical sympathetic from pressure, in bulbar palsy in association with progressive muscular atrophy, in sclerosis of the brain and spinal cord, and in some forms of multiple neuritis.

Mydriasis (dilatation of the pupil). This may be the result of either irritation or paralysis of the centre or fibres governing pupillary activity.

Spastic mydriasis occurs in hyperæmia and irritation of the cervical portion of the sympathetic, in tumors of the cord and brain (although

rarely); in tabes dorsalis; in certain forms of intestinal irritation, especially intestinal tumors; in anæmia; in psychical excitement, for example, fear, surprise, acute mania, melancholia, and progressive paralysis of the insane. In this latter disease the mydriasis is frequently unilateral and is associated with myosis in the other eye.

In spastic mydriasis the pupil is moderately dilated, contracts slightly to light and convergence, and does not dilate to sensory stimuli. Mydriatics dilate the pupil *ad maximum*, but myotics exert but little action upon it.

Paralytic mydriasis, or, as it is sometimes called, *iridoplegia*, is caused by paralysis of the fibres of the oculomotor nerve, the branches which innervate both the intrinsic muscles of the eye—*i. e.*, the sphincter pupillæ and the ciliary muscle—being usually affected. It may be the result of paralysis of the nucleus of this nerve in the pons, or from failure of the stimulus to be conducted from the retina to that centre. The pupil is moderately dilated, reacting to sensory stimuli and to light and on convergence, according to the seat of the lesion. Thus if the lesion be between the iris and the pupil-contracting centre, there is no reaction, either direct or consensual; but if the lesion lie between the retina and the pupil-contracting centre, the pupil will not contract directly to light, although it will consensually and on convergence. Mydriatics dilate the pupil *ad maximum*, but myotics contract it but moderately.

Paralytic mydriasis occurs in diseased processes at the base of the brain, involving the centre of the third nerve; in affections of the orbit which exert pressure on the ciliary nerves; in cerebral processes attended with marked increase in the pressure within the skull, such as tumors, hemorrhages, and abscesses, and in the advanced stages of thrombosis of the cavernous sinus; in progressive paralysis; the later stages of meningo-encephalitis, and acute dementia. Macewen is authority for the statement that hemorrhage into the centrum ovale and cerebral peduncles also produces mydriasis.

Ophthalmoplegia interna is the name given to the dilatation or partial dilatation of the pupil associated with a failure to contract under stimulus; loss of accommodation accompanies it. The condition indicates a nuclear lesion. *Transient mydriasis* affecting first one eye and then the other, is generally regarded as prodromal of insanity.

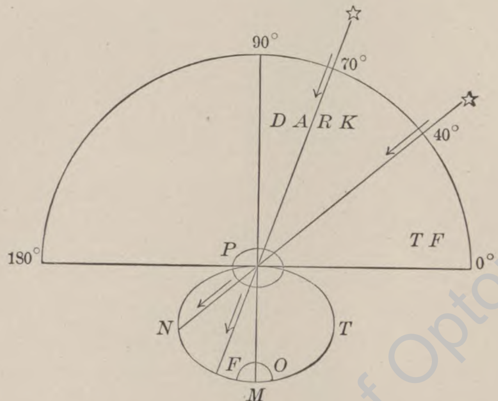
It may be stated as a general rule, that dilatation of the pupil when observed in connection with a cerebral lesion indicates an extensive lesion of the brain; and, when it is of spinal origin, irritation of the part affected. Mydriasis is commonly observed in glaucoma. When mydriasis is due to a failure in transmission of the light stimulus to the pupil-contracting centre and nerves, pupillary activity occurs only on convergence. The mydriasis which accompanies optic atrophy is the type of this class.

The dilatation of the pupil which is observed in complete blindness (*amaurosis*) should not be considered as a disorder in the mobility of

the iris, but should rather be regarded as a physiological inhibition of the pupillary reflex due to the withdrawal of the perception of light.

The hemianopic pupillary inaction sign (*Wernicke's*). By means of this sign it is sometimes possible to determine in certain cases of half-blindness whether the seat of the lesion is situated anterior or posterior to the corpora quadrigemina. (Fig. 9.) This test depends upon the fact that the visual fibres in the optic nerve join the fibres of the third nerve, which control the sphincter pupillæ at the corpora quadrigemina. If the lesion be posterior to this point of junction—*i. e.*, back of the corpora, in the occipital cortex—there will be no interference with the pupillary reflex. Should, however, the lesion be situated anterior to the corpora, the reflex arc of pupillary activity will be broken, and an irregularity in the pupillary contraction will manifest itself. The hemianopic pupillary inaction test is performed best by placing the patient in a dark room, with a single source of

Fig. 9.



Illustrating the test for hemianopic pupillary inaction; the lines represent a horizontal plane through the left eye and its visual field. *F O.* Fundus oculi. *M.* Macula lutea. *N.* Nasal half of the field, which is anæsthetic in temporal hemianopsia. *T.* Temporal half of retina. *T F.* Temporal field. *P.* Pupillary aperture. 180° to 0°, the equatorial arc or semicircle. 90°, vertical point and line passing through centre of eye to *M.* 70° and 40°, rays of light striking the insensitive nasal half of the retina, producing no pupillary reflex. (Seguin.)

illumination back of him, the eye not under examination being closely bandaged, and the patient being directed to look into the distance. An assistant should then moderately illumine the eye by directing light upon it by a plane mirror, while the examiner turns a narrow beam of light, reflected from the concave mirror of his ophthalmoscope, upon the different parts of the retina, and closely observes the effect upon the movements of the iris.

The cerebral cortex reflex of the pupil, or *Haab's reflex*, is the contraction of the pupils in both eyes which occurs without change of accommodation or convergence when the subject, seated in a dark room, directs his attention to some bright object within his field of

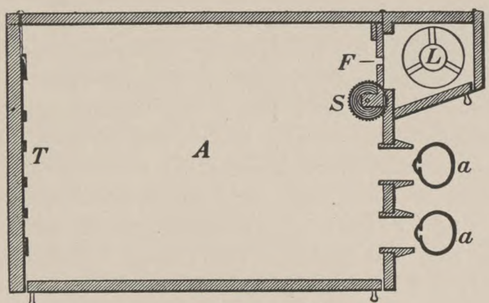
vision, the amount of contraction being proportionate to the brightness of the object.

The orbicularis pupillary reaction, which is used to determine a possible paralysis of the sphincter of the pupil, is a contraction of the pupil which occurs upon forced closure of the lids. Gifford, the discoverer of this reaction, believes this contraction to be an overflow stimulus which is excited in the nuclei of the orbicularis fibres of the facial nerve, and conveyed thence to the pupil-contracting centre. It is believed that when trigeminal anæsthesia is associated with reflex pupillary immobility it may be ascribed to a lesion of the spinal root of the fifth nerve.

Paradoxical pupillary reaction refers to the condition said to be observed at times in meningitis, when the pupil dilates upon exposure to light, and contracts upon its withdrawal.

The Light Sense. In order to test the power possessed by the retina and the visual centre of appreciating variations in the intensity of the source of illumination, an instrument is employed which

FIG. 10.



Photometer of Förster. (FUCHS.)

is known as a *photometer*. (Fig. 10.) This consists of a square box in which are placed black lines equal to certain standard letters when seen at one-third of a metre from the eye. These lines are illuminated by a standard candle, the degree of light being regulated by a window, the size of which may be varied. The patient is first made to look into the apparatus with the window closed. The window is then slowly opened and the lines illuminated. As soon as the lines are recognized the size of the opening requisite for this purpose is noted, and if it be found that an opening more than 2 mm. square has been required, a subnormal light sense is present. Before making this test, it is necessary that the subject under examination should remain in a dark room for at least ten minutes, in order to adapt the retina to the absence of light. The study of the diminution of the light sense is of great value in certain diseases of the retina, when lowering of its acuteness out of proportion to the visual acuity is of great significance.

The light sense of the periphery of the retina may be tested by passing a candle flame affixed to the arc of a perimeter through the

different meridians in front of the eye, while a second candle is employed as a point of fixation. (*Vide* Field of Vision.) Holden's test for this purpose, consists in ascertaining the points on the perimeter at which a series of test objects is perceived. To detect disturbances in the intermediate and central zones of the field, he employs a card with a 1 mm. black point on one side and a 15 mm. quadrant of light gray, having four-fifths of the intensity of the white, on the other. With a perimeter of 30 cm. radius the black point and gray patch are each seen by the normal eye: outward 45 degrees, upward 30 degrees, inward 35 degrees, downward 35 degrees. To detect peripheral defects, he employs a second card, which has a 3 mm. black point on one side, and a darker gray patch having three-fifths the intensity of white on the other. Each should be seen on the perimeter at the following points: outward 70 degrees, upward 45 degrees, inward 55 degrees, and downward 55 degrees.

Tension. As the degree of tension or intra-ocular resistance is liable to vary in many diseased conditions, it is essential that this be ascertained in every case.

Clinically it is not possible to ascertain directly the intra-ocular pressure, but a sufficiently accurate estimate may be made of it by registering the tension of the eyes as felt with the fingers through the upper lid. To do this, it is best to employ the two index fingers, the other fingers being spread out on the temple and brow to afford the hands support. The patient is directed to look down, and slight pressure is made on the globe alternately with the two fingers. The degree of tension depends not only upon the intra-ocular pressure, but also to a slight extent on the elasticity of the sclerotic, which varies with age, and also with the individual. It is important, therefore, in estimating whether the tension of the eye is greater or less than normal, always to compare it with that of the fellow eye, presuming that it is unaltered.

The different degrees of tension are noted as follows:

T. N. Normal tension.

T. full. Slightly more than the average normal tension.

T. + 1. A slight but decided increase above the normal tension.

T. + 2. More marked increase of tension, but where the fingers can still slightly impress the globe.

T. + 3. Increase of tension so marked that no impress can be made on the globe.

Diminished tension in the same way is recorded as of three degrees: T. — 1, T. — 2, T. — 3.

Several different instruments have been invented for recording the tension of the eye, called *tonometers*. None of them is of sufficient practical use to need description here.

THE OPHTHALMOSCOPE.[1]

The student having acquainted himself with the appearance of the exterior of the eye and as much of the interior as may be learned by lateral illumination is now prepared to complete the examination, by exploring minutely the interior of the eye.

On account of certain optical lesions, such an examination cannot be made by the naked eye, and the student will have to call to his aid a device which will overcome these and remove the hinderances which oppose his view into the eye. Although the principles of such a device were suggested and nearly attained by a number of early scientists, the honor of perfecting and elaborating them into an instrument belongs to Helmholtz. This distinguished physicist invented the *ophthalmoscope* in 1851, and by this instrument solved the problem of simultaneously illuminating and viewing the interior of the eye; and although the instrument employed by him was crude and inefficient in many ways, it comprised all of the optical principles of the later-day ophthalmoscope.

The discovery of this instrument immediately opened a new field not only in the study of ophthalmology, but in that of clinical medicine as well. Conditions which were described by the older writers under the names of amaurosis and black cataract, terms used to designate all forms of blindness the cause of which was not apparent from an examination of the anterior segment of the eye, were resolved into various diseases of the optic nerve, retina, and choroid. It was found, furthermore, that systemic disorders quite remote from the eye, such as affections of the kidney, heart, and brain, often presented their first manifestations in the hidden tunics of the eye. Upon this account no routine examination of the eye can be considered to be properly performed until the ophthalmoscope has been employed, nor is a clinician justified in rendering a diagnosis in many cases until he is aware of the intra-ocular condition. It should be remembered that by no other means is it possible *to see* a living nerve head and to study the complete vascular cycle in an organ, of the entrance of its arterial and the exit of its venous blood.

Like most instruments of a similar nature, its use presupposes a certain amount of practice, and the student will succeed in acquiring ability to use the ophthalmoscope only after the exercise of considerable patience and endeavor. One approaching its study should not easily be discouraged, nor should he expect to become expert in its use in a few weeks' time; he should sedulously cultivate every opportunity that presents itself to examine the interiors of healthy eyes, for it is only by a knowledge of the physiological that pathological conditions may be recognized. Artificial eyes, such as Perrin's, are of decided value when it is impossible to obtain natural eyes for

[1] The optical principles involved in ophthalmoscopy, the theory of the ophthalmoscope, and the determination of the refraction of the eye by it will be considered in the chapter on Refraction.

study, or a rabbit's eye may be utilized. In order that the student may be taught accuracy, and may appreciate fully the nature of the changes which the ophthalmoscope reveals to him, it is advisable for him to sketch what he sees; for while but few persons possess sufficient skill to make an artistic drawing of the findings of the ophthalmoscope, nearly everyone may produce a schematic representation of them, especially if he employs a sketch-book such as has been devised by Haab.

The writer knows of no objection to the student's making his first trials with the ophthalmoscope upon eyes the pupils of which have been artificially dilated. The employment of the instrument is greatly facilitated thereby, and the fact that he has once seen the fundus will enable the beginner to proceed with more confidence upon eyes the pupils of which are undilated than he who has never experienced the sensation of actually viewing the head of the optic nerve and the retinal vessels. To avoid any possible accident resulting from the employment of mydriatics, individuals under forty years of age should be chosen, and eyes which are free from external signs of disease. Homatropine in weak solution (4 grs. ad. f℥j) is a convenient drug to employ for this purpose, as it produces a dilatation of the pupil in thirty minutes, and its effects are very evanescent, and may be rendered still more so by the instillation of a few drops of a solution of eserine (eserine sulph., gr. ss; aq. dest., f℥j) at the completion of the examination.

Although not essential, unless the student have markedly defective eyes, it is desirable, to avoid errors in the estimation of the refraction and to obtain the clearest image of the fundus possible, that he have the refraction of his own eyes estimated and corrected, if needs be, by glasses before attempting ophthalmoscopic work.

Method of Ophthalmoscopic Examination. [Note. Since the principles which govern ophthalmoscopy are not described until the next chapter, it will be supposed, to avoid ambiguity, that both the eyes of the surgeon and those of the patient are of normal refraction.]

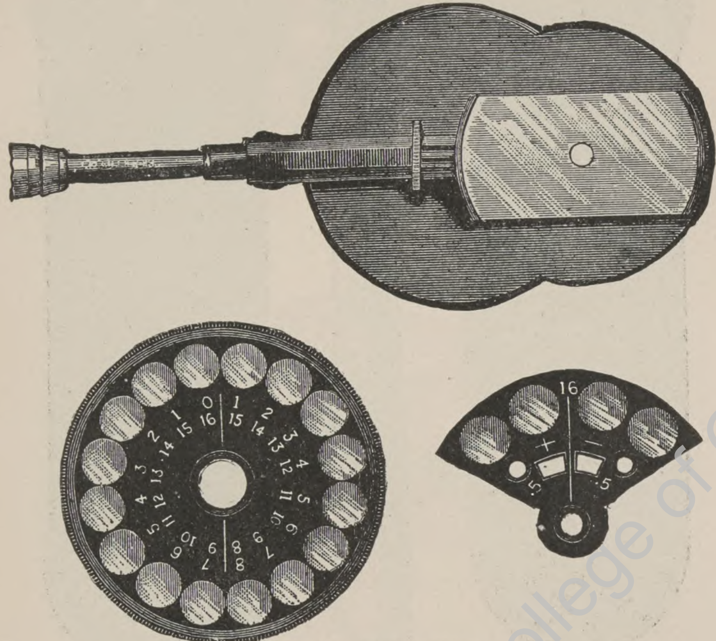
The Instrument. The most essential part of an ophthalmoscope is a mirror with a central perforation, as it is necessary that the light should be reflected into the eye and an opportunity afforded the observer to gain a view of its interior at the same time. There should be two mirrors on every instrument: a plane one to detect opacities in the media and slight changes in the color of the eye-ground, and a concave one of about 30 cm. focal distance, for ordinary examination. In addition to the mirrors, every ophthalmoscope should be provided with a series of lenses to neutralize the refraction of the eye, in order to obtain a sharp and distinct view of the details of the background of eyes of abnormal refraction.

While there are a host of ophthalmoscopes employed throughout the world, the most popular in the United States and the United Kingdom are the Loring and the Morton ophthalmoscopes, respectively. Both of these instruments exhibit to a marked degree the

most essential features in an ophthalmoscope, viz.: largeness of field of view, proper illumination, and the ability to bring a variety of lenses before the sight-hole in the mirror, without the necessity of removing the instrument from the eye. When properly manufactured, either of these instruments should last the average ophthalmologist a lifetime.

The Loring Ophthalmoscope. As shown in Fig. 11, the Loring ophthalmoscope is provided with a concave mirror, parallelogram in shape, with a central perforation from 3½ to 4 mm. in diameter. The mirror is so hung upon the frame that it may be tilted 20 degrees to the right or to the left. For the purpose of focusing

FIG. 11.



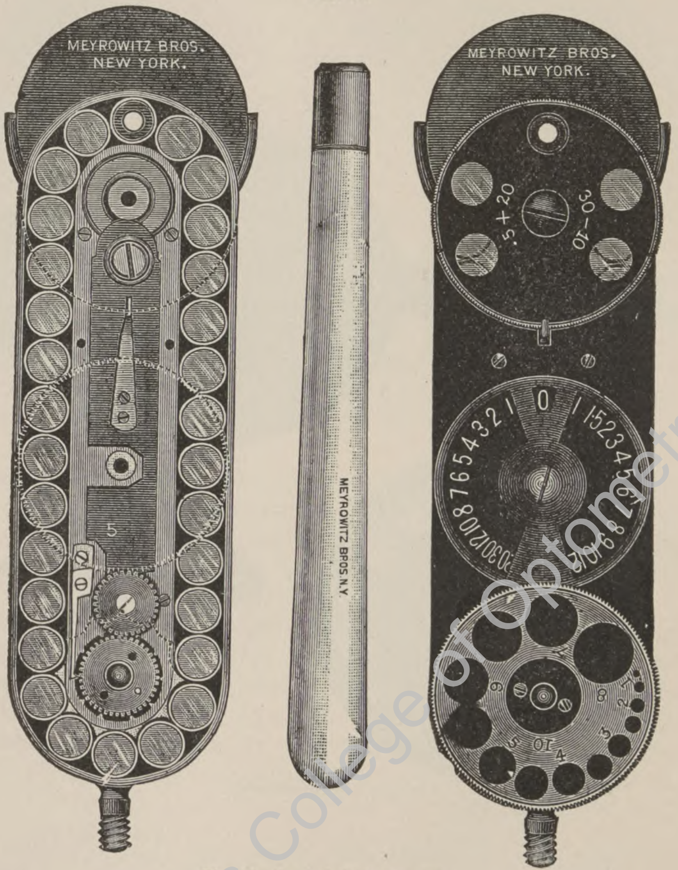
Loring's ophthalmoscope.

the rays upon the fundus in the event that the formation of the eye is abnormal, and secondarily to obtain the degree of refraction error of the eye, the instrument is provided with a series of lenses which may be rotated behind the sight-hole in the mirror. These lenses are contained upon a full disk and a quadrant of a disk, and range from the weakest convex and concave spherical lenses to those of a strength sufficient to neutralize the highest ocular defects.

The Morton Ophthalmoscope. (Fig. 12.) In this instrument the lenses are set in a cylinder in the form of an endless chain, and are propelled by a strong driving-wheel. The instrument is provided with three mirrors—one plane and two concave, one of 10 inches focus and

a smaller one of 3 inches focus. The two first, which are set back to back in one mounting and are reversible, are for indirect examination and retinoscopy; the smaller concave one is for the direct method. Quite recently a stationary ophthalmoscope has been introduced by Thorner (Fig. 13), which, although too large to be manipulated by the hand, and consequently valueless in ordinary routine work, affords an unequalled opportunity of viewing the fundus without annoying

FIG. 12.



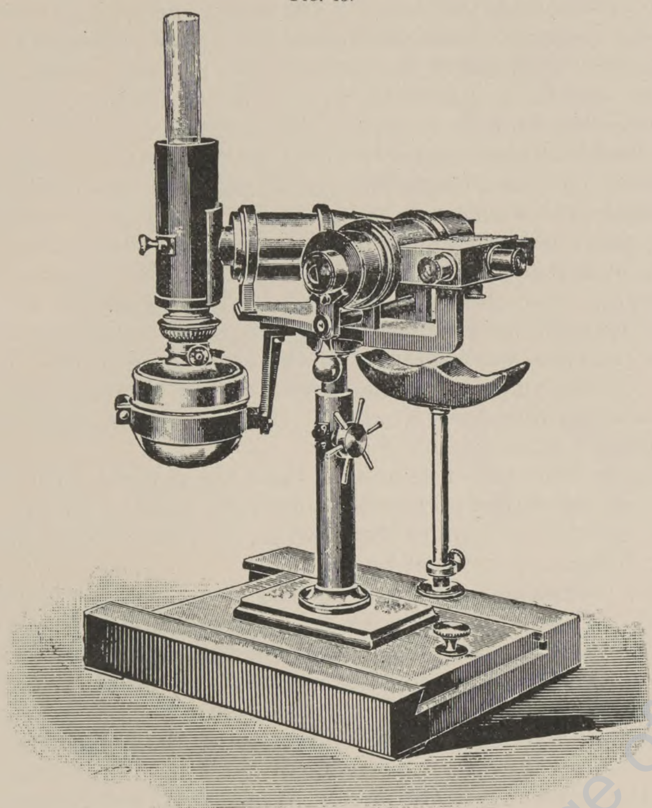
The Morton ophthalmoscope.

reflexes under brilliant illumination and high magnification, and without the necessity of relaxation of the observer's accommodation. By an arrangement of prisms it is possible for the teacher to adjust the instrument for the student, and to demonstrate the changes in the fundus by viewing them simultaneously with him.

Examination by Transmitted Light. Before proceeding to the examination of the details of the background of the eye by either the direct or the indirect method, the student should first acquaint

himself with as much as may be learned by simply throwing the light of the ophthalmoscopic mirror into the eye at a distance of from 30 to 40 cm. For the proper performance of this test, as

FIG. 13.



The Thorner ophthalmoscope.

well as for the methods of ophthalmoscopic examination to be presently mentioned, the patient should be seated in a darkened room, with a light placed a little behind the head and to the side of the eye under examination and upon a line with the ear. An Argand burner

FIG. 14.

Incandescent coil for ophthalmoscopy,

may be employed, or a closely woven incandescent coil (Fig. 14) placed in a bracket which should be capable of being moved in all directions. An old-fashioned oil lamp with a broad flame gives an excellent

light, and daylight may be employed by permitting the rays to gain entrance into the room through a narrow slit in a blind or shutter. Sunlight is to be preferred when it is desirable to study changes in the fundus as nearly as possible in their natural color, as in anæmia.

If the patient be bedridden, it is frequently necessary to resort to a candle as a source of illumination, and the observer may be compelled to make the examination in a constrained posture. Such examinations are apt to be unsatisfactory, but the student should never desist nor allow himself to become discouraged until he has satisfied himself that he has accomplished all that was possible under the circumstances. In the performance of all ordinary tests with the ophthalmoscope, the observer should be on a slightly higher level than the patient, and both he and the patient should be comfortably seated; the custom which prevails in some places of the surgeon standing and bending over the patient's eye is deprecated as tending to favor hurried and superficial examinations. In examining children's eyes the patience and ingenuity of the observer will often be taxed before a satisfactory view of the fundus can be obtained, and a third person is often necessary to attract the gaze of the child from the mirror toward some distant object.

In examination with transmitted light, the student, with the large concave mirror of the ophthalmoscope held close to his eye, throws the light upon the eye under observation, the patient being instructed to direct his gaze in front of him.

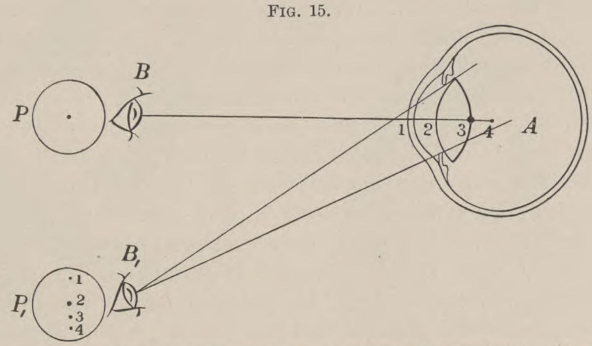
A faint pinkish-red glow will be seen to replace the blackness of the pupil. This is known as the *fundus reflex*, and is occasioned by the reflection of light from some part of the illuminated interior of the eye. With the light from the mirror still concentrated upon the pupil, the student should now tilt the mirror in different directions and note the character and the direction of the movements of the shadow which will be seen to travel over the eye, thus obtaining an idea of the refraction. The patient may then be requested to rotate his eye through the different meridians, and following this the student should move his own head from side to side and back and forth, the better to obtain the reflex from all parts of the eye. The mobility of the iris should be tested by throwing the light upon the pupil, and the perceptive power of the entire retina ascertained by reflecting the light from different angles upon all parts of that membrane.

By the use of the mirror it is also possible to determine *the fixation point*. This is done by observing the corneal reflex. This method, attention to which was called by Priestley Smith, is practised in the following way: the patient is told to look at the mirror; the light is then thrown upon one of the patient's eyes, and the exact position of the light reflex upon the surface of the cornea noted; the student then quickly turns the light to the other eye and compares the position of the corneal light reflex in the two eyes. The corneal reflex generally appears a little nearer the inner than the outer edge of the pupil, as the visual axis usually lies to the inner side of the axis of

the cornea. If both eyes be properly directed, the position of the corneal reflex will be symmetrical in the two eyes; but if one eye deviates, the reflex will be displaced. In this way imperfect fixation in strabismus will be readily detected.

By transmitted light alone, the presence of *opacities in the media* may be diagnosed; these appearing like dark shadows in the red background, because the rays of light as they return from the eye-ground are arrested by the opaque spots in the media, just as all objects which do not transmit light appear dark when seen in front of a luminous surface. As they often are seen best with feeble illumination, it is well to substitute the plane mirror for the concave in searching for them. With a view to examining the media more closely, and to ascertain more definitely the character and position of any opacities, the student should now rotate a high convex spherical lens before the sight-hole in the ophthalmoscope and approach the eye until he is within the focal distance of the lens. For this purpose,

FIG. 15.



Diagnosis of the site of an opacity from parallactic displacement. (FUCHS.)

the Morton ophthalmoscope is provided with a lens of 5 cm. focal length (+20 D.), the Loring with one of 6.25 cm. (+16 D.). Under this high magnification any foreign body or opacity which may have been overlooked usually becomes visible. To determine the exact location of opacities is often difficult, and careful observation is required to state definitely whether they be in the cornea, in the anterior portion of the lens, in the posterior portion of the lens, or in the anterior portion of the vitreous. It may, however, be stated as a general rule, that stationary opacities are in the cornea and lens, and that opacities in the vitreous, although at times fixed, are usually floating. It is frequently possible also to determine the location of an opacity by comparing its position with other structures in the eye in the same plane, as, for example, the conjunctiva and the limbus in cases of opacity of the cornea, and the iris with the anterior part of the lens.

A very accurate, and at the same time a very simple mode of locating the position of an opacity is by means of the *parallactic displacement* of the opacity with reference to the margin of the pupil. In

Fig. 15, 1, 2, 3, 4 represent four opaque points in the optical axis of the eye, situated in the cornea, upon the anterior capsule of the lens, at the posterior pole of the lens, and in the anterior part of the vitreous, respectively. When the observer is stationed at B, all four points will be merged, and he will see but one. Should, however, he move to B, then the position of these points in relation to the pupil will be changed. Thus, 2 will remain fixed, while 1 approaches the upper, and 3 and 4 the lower part of the pupil, 4 the more so. In the application of this test, the observer notes the position of the opacity by looking directly into the eye along its optical axis. He now slowly moves his head to one side. If the spot remains immobile, it is situated in the plane of the pupil. If it moves in a direction opposed to that of the observer's eye, the opacity is situated anterior to the pupillary plane. If the motion is in the same direction, then the opacity is situated in the deeper part of the lens or in the anterior portion of the vitreous.

Having completed this preliminary study of the media with the mirror and by the use of the high magnifying lens, the student should now rotate the disk upon the ophthalmoscope until the sight-hole of the instrument is once more unobstructed by a lens, and should then proceed to an examination of the eye-ground itself. For this purpose he has the choice of two methods, the direct and the indirect; though the beginner will do well to familiarize himself with both in all cases.

Indirect Method. In the application of this method, the student stations himself in a position corresponding exactly to that assumed in the test by transmitted light, at a distance of about 30 cm., and throws the light into the eye under examination by the concave mirror of the ophthalmoscope. As has just been described, the red reflex of the fundus will at once become visible; but unless the eye be highly nearsighted, nothing more will be observed unless a convex lens of about 5 cm. focus be interposed before the eye and held at its focal distance. If this be done, an inverted image of the eye-ground is obtained, which will be seen between the lens and the student's eye. Some difficulty is usually found by the beginner in accomplishing this, on account of reflections from the surface of the lens and the cornea, and his tendency to accommodate either for the eye or the auxiliary lens. The reflections may be overcome by gently tilting the lens from side to side, by bearing in mind also that the image of the fundus is an aërial one, and by making an attempt to adjust the eyes, both being kept open, upon a point between his own eye and the lens; the latter difficulty may be dispelled also, and he will avoid the natural tendency to accommodation. In examining the eye, the student should apply his right eye to the sight-hole in the mirror, the instrument being grasped by the right hand while the auxiliary lens is held in the left. It is advisable to steady the hand which holds the auxiliary lens by resting the tip of the little or ring finger upon the patient's brow, and to utilize one of these fingers to raise the upper lid when it is desirable to examine the lower part

of the eye, or if the lid is covering the pupil, as is frequently the case in inflamed eyes.

If the right eye is under examination, in order to bring the head of the optic nerve into view the patient should be told to direct his gaze at the raised little finger of the observer's right hand as it grasps the tip of the handle of the ophthalmoscope. When the left eye is being examined, he should look at the observer's left ear.

The indirect method is to be preferred when it is desirable to obtain a general view of the fundus and in eyes with hazy media or in high myopia, as the image obtained by it is more luminous than that from

FIG. 16.

The indirect method of ophthalmoscopic examination.

the direct method. By this method the image of the fundus is magnified about five diameters, ten diameters less than by the direct method, but greater magnification may be obtained of the aërial image by rotating a + S 4 D. lens before the sight-hole of the ophthalmoscopic mirror.

It should be remembered that the image in the indirect method is an inverted one, and that, therefore, the upper part of the image corresponds to the lower part of the eye-ground, and the right side of the image to the left of the eye-ground. It is extremely useful in examining patients in a recumbent posture, and is very valuable in examining the eyes of children, as it is often impossible to obtain a

view of the fundus in this class of cases by the direct method, on account of the impossibility of keeping them quiet.

On account of its greater magnifying power, the direct method is to be preferred for the recognition of fine details in the fundus, and it possesses the further advantage over the indirect in that it is possible to estimate the refraction of the eye by it.

The Direct Method. In the application of this method the student approaches as closely to the face of the patient as is possible until the ophthalmoscope is brought within one inch of the patient's eye. The instrument should be grasped with the right hand when the right

FIG. 17.

The direct method of ophthalmoscopic examination.

eye is being examined; but when the left eye is under examination, the hand as well as the position of the light should be changed. The student should aim to keep the pupil steadily illuminated, and should endeavor to keep the small circular shadow which occupies the centre of the glare thrown by the mirror, and which represents the sight-hole of the ophthalmoscope, directly in the centre of the pupil. This he will find greatly facilitated by the practice he has acquired in the observation of the eye by transmitted light and in the indirect method. The red reflex of the fundus should at once become plainly visible; but, as a rule, nothing more, the details of the fundus being still hidden from view. This may be due to

reflections from the cornea and lens, which are especially disturbing in those with deeply pigmented irides and small pupils, or to an active accommodation in either the surgeon's or the patient's eye. To decrease these reflections, the mirror should be moved almost imperceptibly from side to side until a spot is found where the reflections seem to disappear. For optical reasons which will be explained in the next chapter, it is necessary, in order to see the details of the fundus clearly, that the accommodation in both the observed and the observing eye should be relaxed. This is accomplished in the patient's eye by having him look into the distance in a darkened room. Practice alone, however, will enable the student to lose the desire to accommodate, which has been natural to him in regarding all near objects hitherto, and to view the interior of the eye situated but an inch or so from him as though it were a far-away object. He will find that relaxation of the accommodation will be facilitated greatly by keeping both eyes open, and this should be practised during the entire test, for while the images formed upon the unemployed eye will at first be confusing, he will soon accustom himself to ignore them.

The student should now search for the head of the optic nerve, as this is the most prominent feature in the fundus. To bring this into view, he should request the patient to direct his gaze slightly toward the left when the right eye is being examined, and *vice versa* for the left eye. If he now looks closely, he will observe that the glare from the fundus is not uniformly red, but that it contains a disk of color which is yellowish white. This whitish reflex should be kept steadily in view, and as his accommodation relaxes he will find that the whitish disk resolves itself into an oval body yellowish white in color, forming a strong contrast to the reddish color of its surroundings. This is the head of the optic nerve. If the nerve does not come into view, one of the retinal vessels frequently will, and this should be followed up until the nerve is reached, the avenue of entrance and exit of the retinal circulation.

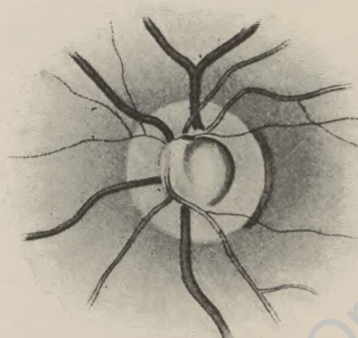
Let no one fancy, however, that the interior of the eye reveals itself at the first attempt of the beginner, nor let him be discouraged on that account, for usually much persistence and no little pains will be demanded of him before he accomplishes his purpose. It is not desirable at first for the student to try to discover the lens in the ophthalmoscope with which he sees the details of the fundus best, for this is only possible after much practice and after he has learned to relax his accommodation thoroughly. For determination of the refraction by the direct method, *vide* page 118.

The Normal Eye-ground. (Plate II.) On account of the many variations in the normal eye-ground, the beginner is urged to familiarize himself with the appearance of many fundi which are known to be healthy, to prevent confounding physiological changes with pathological ones. He will find the study of children's eyes especially well adapted for this purpose, as their pupils are likely to be larger

and the refracting media clearer than in older people, and, as a rule, they submit more willingly to the irksome routine of the examination.

The most prominent feature in the back part of the eye, or *the fundus*, is the head of the optic nerve, *the optic disk*, or, as it is often called, by reason of its slight elevation in some cases beyond the surrounding retina, *the optic papilla*. Reddish yellow in color, with the retinal vessels tapering from it, it forms a striking contrast with the uniform reddish coloration of the rest of the fundus. Although the general appearance of the nerve is reddish yellow, close inspection will reveal that its nasal portion is slightly darker than the temporal, and that a whitish circle occupies the central or temporal portion of the nerve. This whitish circle represents *an excavation* or cup in the nerve, and is occasioned by the separation of the nerve fibres by the entrance of the retinal vessels into the eye. (Fig. 18.) This cup is usually funnel-shaped, but varies

FIG. 18.



Physiological examination. (SZILI.)

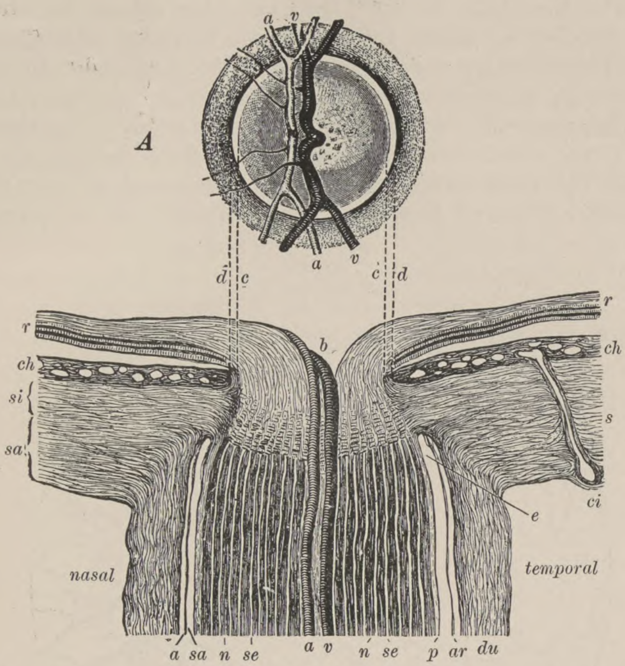
greatly in size and position as well as in configuration. It may be but a small depression marking the entrance of the bloodvessels, or it may embrace the greater part of the head of the nerve. At the bottom of the excavation *the lamina cribrosa* is seen as a whitish plate studded with grayish-black spots, the points of entrance of the nerve fibres.

Although the disk is in reality but 1.5 mm. in diameter, it appears quite large by the direct method, and as it is the only portion of the fundus which has a fixed form, it is used as a landmark to note the size and the position of changes in the fundus, as, for example, a hemorrhage is noted as being in the retina two disk diameters above the disk

Surrounding the optic nerve are two rings of more or less completeness: the innermost, a whitish circle, *the scleral ring*, and bordering upon this a pigmented ring, *the choroidal ring*, as seen in the

accompanying illustration (Fig. 19), which is taken from Fuchs. The scleral ring is the result of a larger opening in the choroid than

FIG. 19.



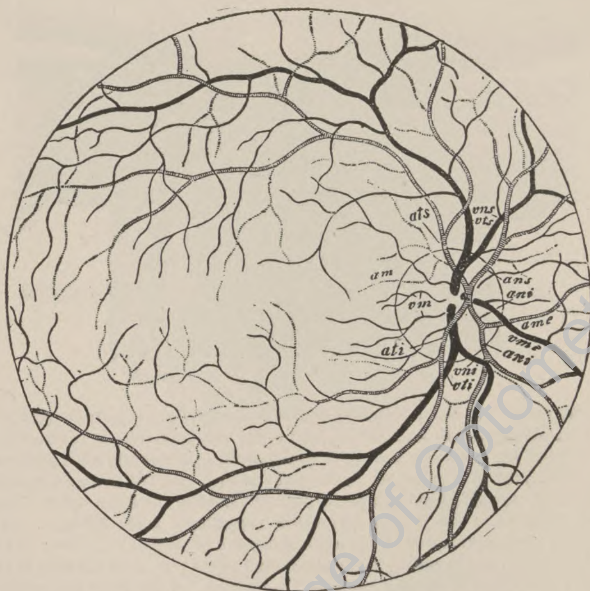
Head of the optic nerve. *A.* Ophthalmoscopic view. Somewhat to the inner side of the centre of the papilla the central artery rises from below, and to the temporal side of it rises the central vein. To the temporal side of the latter lies the small physiological excavation with gray stippling of the lamina cribrosa. The papilla is encircled by the light scleral ring (between *c* and *d*) and the dark choroidal ring at *d*. *B.* Longitudinal section through the head of the optic nerve. Magnified 14 x 1. The trunk of the nerve up to the lamina cribrosa of medullated nerve fibres, *n*. The clear interspaces, *se*, separating them correspond to the septa composed of connective tissue. The nerve trunk is enveloped by the sheath of pia mater, *p*, the arachnoid sheath, *ar*, and the sheath of dura mater, *du*. There is a free interspace remaining between the sheaths, consisting of the subdural space, *sd*, and the subarachnoid space, *sa*. Both spaces have a blind ending in the sclera at *e*. The sheath of dura mater passes into the external layers, *sa*, of the sclera, the sheath of pia mater into the internal layers, *si*, which latter extend as the lamina cribrosa transversely across the course of the optic nerve. The nerve is represented in front of the lamina as of light color, because here it consists of non-medullated and hence transparent nerve fibres. The optic nerve spreads out upon the retina, *r*, in such a way that at its centre there is produced a funnel-shaped depression, the vascular funnel, *b*, on whose inner wall the central artery, *a*, and the central vein, *v*, ascend. The choroid, *ch*, shows a transverse section of its numerous bloodvessels, and toward the retina a dark line, the pigment epithelium; next the margin of the foramen for the optic nerve and corresponding to the situation of the choroidal ring the choroid is more darkly pigmented. *ci* is a posterior short ciliary artery which reaches the choroid through the sclera. The posterior portion of the scleral canal forms a funnel directed backward, the anterior portion a funnel directed forward. The wall of the anterior funnel when seen in front appears to have the extent, *cd*, and corresponds to the scleral ring visible by the ophthalmoscope. (FUCHS.)

the sclera, to permit of the entrance of the optic nerve into the eye, as a consequence of which a portion of the sclera is exposed. The

choroidal ring is formed by the heaping up of pigment where the choroid adjoins the optic nerve.

The vessels of the retina (Fig. 20) consist of a main arterial and a venous stem, the central artery and vein of the retina, which divide, directly after they have emerged from the nasal side of the excavation upon the head of the nerve, into two main branches, the superior and inferior. These further subdivide several disk diameters distant from the disk into the superior and inferior temporal and the nasal branches, respectively, and still further subdivide into smaller branches; these branches never anastomose. (*Vide* page 418.) The macular region is supplied by small twigs from the superior and inferior temporal branches, and often by two small twigs directly off the parent stem

Fig. 20.



Distribution of retinal vessels. (Jaeger.)

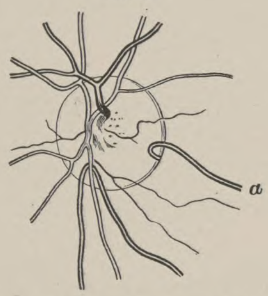
on the disk, the superior and inferior macular arteries; larger vascular stems never invade its territory. The arteries are smaller and straighter than the veins, and are yellowish red, while the veins are purplish. Both have a light streak along the centre of each, which is fainter upon the veins, *the reflex streak*. The veins usually accompany the arteries, and have the same distribution and name. It is usual for a distinct *pulse* to occur in the veins upon the head of the nerve. Pulsation in the arteries, however, is always pathological. The retinal vessels frequently present great variations, both in distribution and characteristics, and the observer will often be puzzled to decide whether such variations fall within physiological limits. The two most striking variations are an artery, which is occasionally seen,

and which arises in the choroid and runs inward toward the disk, then taking a general direction toward the macula, and a *cilio-retinal vessel.* (Fig. 21.) *Optico-ciliary vessel* is the name given to a branch of the central vein or artery which disappears at the edge of the disk.

The student should acquire the habit of observing the retinal bloodvessels with great care, for their condition is frequently indicative of the condition of the bloodvessels elsewhere throughout the system; and on account of the ability actually to view the blood column itself, an exceptional opportunity is afforded him of diagnosing pathological conditions of the blood.

The reddish appearance of the fundus surrounding the optic nerve is due chiefly to the blood in the choroidal capillaries, although the retinal circulation is also a slight factor. Of more influence in affecting the general coloration of the fundus is the pigment in the retina, according as it is present in greater or lesser quantity. Thus in brunettes, in whom there is an abundance of pigment, the general tone

FIG. 21.



Cilio-retinal artery. From the outer and lower margin of the papilla rises a cilio-retinal artery, *a*, making a hook-like bend. In this case it is larger than usual, because it is destined to replace the main infero-external (inferior temporal branch) of the central artery, which branch is wanting. (FUCHS.)

is deep red or even slate color in pronounced cases. In blondes it is often a delicate pink, and, owing to the absence of pigment in the retina and choroid, the entire circulation of the choroid, which is quite obscure in brunette eyes, is laid bare to the gaze. *Albinism* presents this to an extreme degree, the red choroidal vessels being seen to course over the white background that is formed by the sclera. (Fig. 22.) Although the retina in health is transparent at times, especially in children, it is so rich in connective tissue that a striated, grayish appearance is given to the fundus, especially in the neighborhood of the disk. These *reflexes* frequently accompany the vessels, and are so prominent that they give the retina a watered-silk appearance. They are usually more marked around the yellow spot and occasion the halo which surrounds that area.

A number of small shining bright dots are sometimes seen in the retina anterior to the retinal vessels; they were called by Marcus Gunn, who first described them, "*creek dots.*" They are only visible by the

direct method, and are not easily seen. Their nature is not known; they may occur in several members of a family, and are often hereditary.

The macula, the region of greatest importance of the retina, reveals itself only after some difficulty upon the part of the ophthalmologist, as it has no characteristic sufficiently striking to delineate it. It is an oval area, with the long axis horizontal, is more deeply pigmented than the surrounding fundus, and is avascular. From its centre a bright reflex is emitted, *the fovea centralis.*

FIG. 22.

Ophthalmoscopic representation of the eye-ground of an albino. (JAEGER.)

To examine this region when the pupil is undilated, the ophthalmoscope should be slowly moved upward and inward, while the observer brings his line of vision to a point about two to two-and-a-half disk diameters outward from the disk. When the pupil is dilated, the macula may be seen by having the patient gaze directly into the sight-hole of the ophthalmoscopic mirror. The *periphery* of the eye-ground should also be carefully studied; and in order that no part of it may escape, it is well for the student to follow each branch of the central artery of the retina as far forward as is possible.

CHAPTER II.

THE PHYSIOLOGY OF VISION.

BY WILLIAM NORWOOD SUTER, M.D.

VISION is the mental interpretation of an impulse conducted from the rods and cones of the retina through the optic nerves and tracts to the *visual areas* of the brain. These areas are situated in the cuneal and occipital lobes at the internal and posterior region of each hemisphere. As to the manner in which the physical impulse is transformed into vision, we have no knowledge, as we have not of any other kind of perception.

The visual impulse normally results from the action of light on the rods and cones. This action is, in part at least, chemical, the visual purple of the retina being changed into a colorless substance.

Artificial (electrical) stimulation of the optic nerve or of the visual areas causes only the sensation of light (illumination) as distinguished from darkness. For the distinction of objects by the visual sense, it is requisite that the object be reproduced in an image on the retina, thus stimulating only such rods and cones as are covered by the image. In this way a mental picture is realized corresponding to the image delineated on the retina. Thus the question of the physiology of vision resolves itself largely into an investigation of the laws of light, in adaptation to which the eye is constructed.

OPTICS.

Light is a form of energy capable of giving rise to vision, but capable, also, under suitable conditions, of being transformed into other kind of energy.

That branch of science which treats of the laws of light is called *optics*. Optics deals not only with light in its relation to the organ of vision; it investigates the laws which govern light-energy, irrespective of the eye—the organ by which alone the phenomena of light are manifested to our consciousness. It behooves us to consider here, however, only so much of the subject of optics as will afford a correct understanding of the formation of the retinal image.

A body whose constitution is such as to produce light-energy—to emit light—is said to be self-luminous. Such a body emits light in all directions and in rhythmical impulses or waves.

Since we cannot conceive that light or any other form of energy may traverse space without the intervention of a medium, it is neces-

sary to assume the existence of an all-pervading substance, called *ether*, by means of which light-vibrations are transmitted.

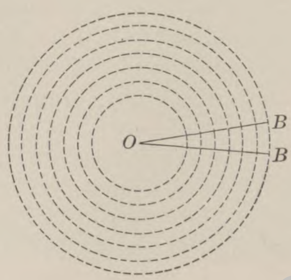
The velocity of light through space is, as demonstrated by astronomical observations, about 300,000,000 metres (186,000 miles) per second.

A luminous body does not ordinarily emit a single wave, but a number of waves of different length and rapidity of vibration. Only those waves within certain limits of periodicity (from 394 million millions to 763 million millions of vibrations per second) affect the eye as vision.

Color depends upon the wave-length and rapidity of vibration The wave of greatest length and least rapidity gives rise to the sensation of red; that of least length and greatest rapidity gives the sensation of violet. Between these limits are comprised all light-waves, which produce the colors of the rainbow or *spectrum*. Ordinary white light is composed of all these waves acting upon the retina in unison.

It is not definitely known how color-sensations are produced; but according to the Young-Helmholtz theory (the commonly accepted

FIG. 23.



one) there are three sets of rods and cones, each set being affected by waves of certain lengths only. These groups of waves correspond to the three primary colors, red, green, and blue. By the combined effect in varying proportion upon the retina of these three elements all color-sensations are produced.

A substance which permits the passage of light is called a *medium* or a transparent body. One which does not permit the passage of light is said to be *opaque*.

When light meets an opaque body, it is either *reflected* back into the medium from which it came, or it is *absorbed*—converted into other form of energy.

If the medium surrounding a luminous point is homogeneous, the light emitted from this point will travel equally in all directions and the wave-front will be spherical. (Fig. 23.)

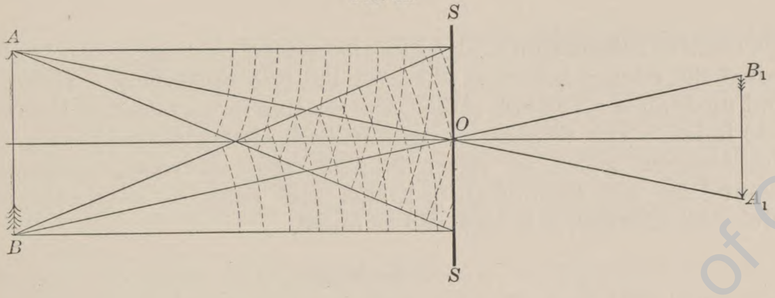
A small portion of this wave (*B O B*), such as might enter an eye, is called a *pencil*. An infinitesimal pencil is called a *ray*. Thus, mathematically, a ray is a straight line (*B O*) perpendicular to the wave-front.

In the study of optics it is often convenient to regard light as composed of rays proceeding in every direction from a luminous point; and we may with propriety make this assumption, provided we do so with the understanding that it does not represent the true mode of transmission.

Every point of a luminous body emits light, hence there proceeds from a body of appreciable size a great number of waves whose paths cross each other in various directions. We must assume, therefore, that many waves may traverse the same medium at the same time, each wave-disturbance being superposed upon that of the other, a principle with which we are familiar in the superposition of motions.

Formation of Images. In order that an object may be reproduced in an image, it is essential that light from any point of the object shall reach a corresponding point on the intercepting screen, and that light from all other parts of the object shall be excluded from this point. The simplest way in which this can be accomplished is illustrated in Fig. 24. *S S* represents an opaque diaphragm in which there is a

Fig. 24.



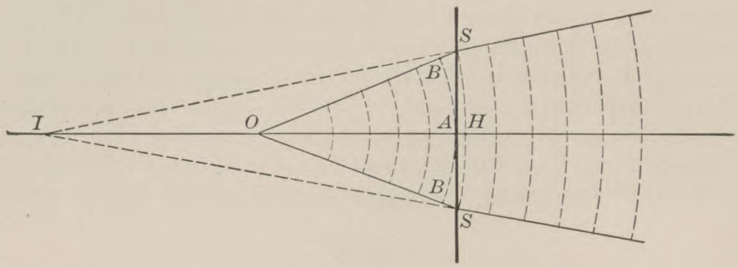
minute opening, *O*. Light from *A* passes through the opening and falls upon the screen at A_1. Light from other parts of *A B* cannot reach A_1. Hence at A_1 the luminous point *A* is reproduced; so for every other point of *A B*, and A_1 B_1 is an inverted image of *A B*. The objection to this device is that the opening must be so minute as to allow the passage of a single ray, or at least of a very small pencil from each point of the object; consequently the image is very feebly illuminated.

Refraction. In the eye and in other optical apparatus greater illumination is secured and the apparatus made more sensitive by refraction of the pencils. By this means, larger pencils are concentrated to a point in the image.

It is found that the velocity of light is less in dense than in rare substances. The effect which this retardation has upon rays is illustrated in Fig. 25, in which *B O B* represents a section of a spherical wave meeting a denser medium in the plane *S S*. That part of the wave which travels along *O A* meets the surface sooner than that which travels along *O B;* hence when the former traverses the distance

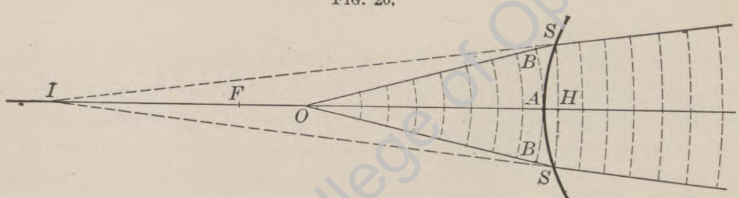
$A\ H$ in the dense medium, the latter traverses the greater distance $B\ S$, in the rare medium. In this way the wave-front is flattened so that the centre of the wave is now situated at I.[1] $S\ H\ S$ being the wave-front, the rays are represented by lines drawn perpendicular to the arc $S\ H\ S$, as $I\ S$ and $I\ H$. The direction of all the rays is changed except that of $O\ A$, which is perpendicular to the surface. All other rays are said to be *refracted.* The degree of refraction depends upon the obliquity with which the rays meet the surface and

FIG. 25.



the relative retardation of light by the second medium. The latter is called the *relative refractive index* for the two substances. When the first medium is a vacuum (ether) the relative index becomes the absolute index. The velocity of light *in vacuo* being regarded as unity, the absolute index of water is 1.33, that of spectacle glass is about 1.52, and that of air is 1.0003, and, being so nearly identical with that of ether, it is regarded as unity.

FIG. 26.



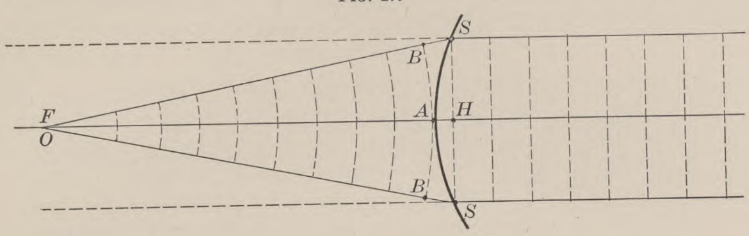
Refraction at Curved Surfaces. What has been illustrated as occurring at a plane surface occurs similarly at a curved surface. Fig. 26 illustrates the passage of a wave from a rarer to a denser medium at a convex surface. In this case the flattening of the wave is greater than it would be as affected by a plane surface; but, as with the plane surface, the pencil is still divergent after the refraction. With greater convexity or refractive index the flattening may be such that the wave is plane after refraction, as illustrated in Fig. 27; the refracted rays are parallel.

[1] We assume for the present that the refracted wave-front is spherical, and that the refracted rays all proceed from the same point, I; we shall learn later that this assumption is permissible only when a small portion or pencil of the wave is considered.

Thirdly, as illustrated in Fig. 28, the retardation may be so great that after refraction the rays converge to a point, *I*, which is the *focus* of the refracted pencil. This focus is illuminated by all the rays of the pencil *S O S*; it is consequently a bright point corresponding to the bright point *O*, from which the pencil proceeds. *I* is the image of *O*, and the two points are called *conjugate foci* with respect to each other.

In Fig. 26 light from *O* appears after refraction to come from *I*; *O* and *I* are, as in Fig. 28, conjugate foci, but in this case *I* is not an

FIG. 27.



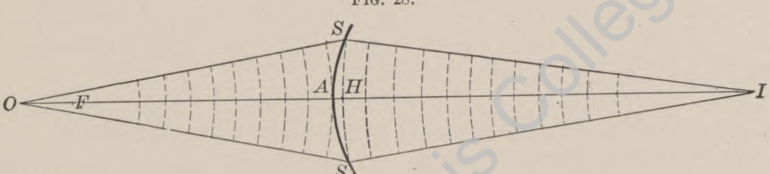
illuminated point. It is called an imaginary or *virtual* focus, in contradistinction to the *real* focus *I* in Fig. 28.

The distances *O A* and *I A* are called *conjugate focal distances*; the line *O I*, on which the distances are measured, is called the *axis*.

In Fig. 27 the rays are parallel to the axis after refraction, that is, mathematically they intersect the axis at infinity. The point *F*, so situated that the rays are parallel after refraction, is called the *principal* focus.

Relative Positions of Conjugate Foci. If we examine mathematically the relation between conjugate foci, we find that when *O* is

FIG. 28.



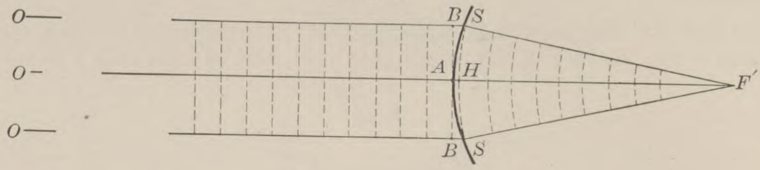
situated nearer the refracting surface than the principal focus the conjugate focus *I* is virtual; it lies on the same side of the surface as *O*. (Fig. 26.)

When *O* is more remote from the surface than the principal focus, the conjugate *I* lies on the opposite side of the surface, and is real. (Fig. 28.)

As the point *O* recedes from the surface the conjugate on the opposite side approaches the surface, and when the distance *O A* becomes infinite, that is, when the incident wave becomes plane (the rays

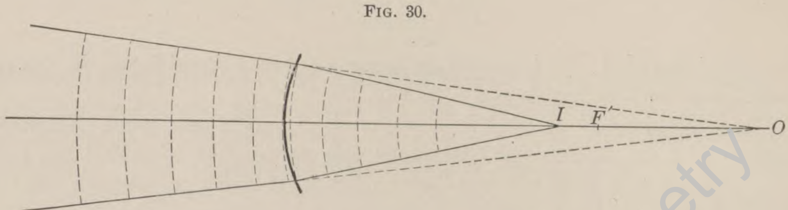
parallel), the refracted wave converges to the point F' (Fig. 29), which is the *posterior* principal focus, in contradistinction to F (Fig. 28), which is the *anterior* principal focus. Rays proceeding from the anterior principal focus are parallel *after* refraction, and rays which are parallel *before* refraction converge to the posterior principal focus.

FIG. 29.



Fourthly, the point O may lie to the right of the surface, that is, the wave is already converging to this virtual focus before refraction. In this case I lies on the same side of the surface as O and nearer to the surface. (Fig. 30.)

FIG. 30.



Collective Refraction. In the condition illustrated in Fig. 26 the divergence of the pencil is diminished by the refraction; in that illustrated in Fig. 27 the divergence is neutralized; in the condition illustrated in Fig. 28 the divergence is more than neutralized, the wave is rendered convergent, and in the fourth condition the convergence of the already converging pencil is increased. Hence the refraction which occurs when light passes from a rarer to a denser medium at a convex surface is *collective* or *convergent*.[1]

It is evident that we may reverse the course of light in these illustrations, that is, we may regard I as the focus before refraction, and O as the conjugate after refraction. Hence these diagrams serve equally well to illustrate refraction which takes place when light passes from a denser to a rarer medium at a concave surface. Such refraction is therefore collective.

Dispersive Refraction. It would be superfluous to illustrate here the refraction which occurs when light passes from a rarer to a denser medium at a concave surface, or, its equivalent, that at a con-

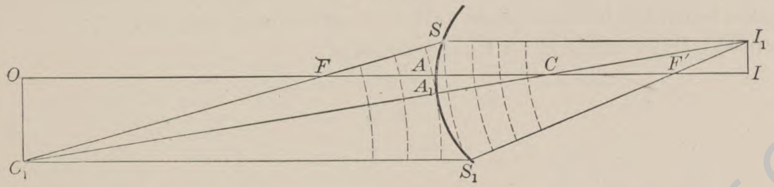
[1] An exception to this occurs: (1) when the incident wave is directed toward the centre of the surface; all the rays then being perpendicular to the surface there will be no refraction; and (2) when the wave is converging to a point to the left of C, that is, to a point nearer the surface than the centre, in which case the divergence of the pencil will be increased—conditions which do not arise in ocular refraction.

vex surface when light passes from a denser to a rarer medium. It is apparent that the effect of such refraction is opposite to that which has been illustrated, that is, the divergence of the pencil will be increased by such refraction. This is called *dispersive* refraction. A pencil of light diverging from a point, being rendered still more divergent by dispersive refraction, can never be united by such in a real focus.

Formation of Images by Collective Refraction. The formation of images by refraction is illustrated in Fig. 31. All rays of the pencil diverging from O_1 are concentrated at the conjugate focus I_1. No light from other parts of the object $O\ O_1$ can reach I_1, but each point lying between O and O_1 has a corresponding conjugate lying between I and I_1; hence $I\ I_1$ is the image of $O\ O_1$.

We have learned that in collective refraction there will be a real focus conjugate to any point (O_1) when this point is further from the surface than the first principal focus; hence there will be a real and an inverted image of $O\ O_1$ whenever the distance $A\ O$ (or $A_1\ O_1$, for $O\ O_1$ and $I\ I_1$ are really arcs of circles whose radii are $C\ O$ and $C\ I$) is greater than the principal focal distance $A\ F$.

FIG. 31.



Cardinal Points. It is apparent that any ray, as $O_1\ I_1$, which passes through the centre of curvature of the refracting surface undergoes no refraction. From this property the point C is called the *nodal point;* and any ray ($O_1\ I_1$) passing through this point is called a secondary axis, in contradistinction to the primary or *principal* axis $O\ I$. The point A, where the surface intersects the principal axis, is called the *principal point.* These two—the nodal and principal points—together with the two principal foci constitute the *cardinal points* of the refraction.

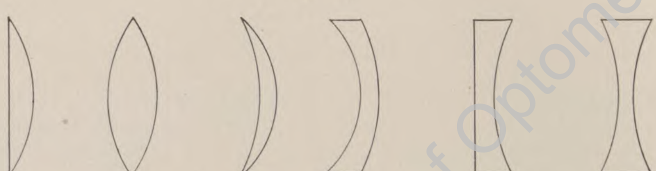
If, as has been done in the figure, the diagram be so drawn that the incident ray $O_1\ S_1$, and the refracted ray $S\ I_1$, are each parallel to the axis $O\ I$, it is evident that in so doing we have a means of determining the position of the principal foci F and F'. Conversely, if we know the position of these foci and of the other two cardinal points, we may by the same geometrical construction ascertain the position and the size of the image ($I\ I_1$) of an object, $O\ O_1$.

The Aphakic Eye. Since it is requisite for vision that a real image of the object viewed be formed on the retina, it is apparent that the eye must be so constructed as to constitute a collective refractive apparatus. The simplest device of this kind is that of a single surface,

just illustrated. The first medium is the air, through which light from external objects is normally conducted to the eye. The refracting surface is the anterior surface of the cornea, a convex surface separating the air from the denser interior substance of the eye, namely, the cornea, the aqueous humor, and the vitreous body, the refractive index of all these media being practically identical.[1] This is the condition of the eye deprived of its crystalline lens—*the aphakic eye.* Such an eye, provided it has sufficient length, fulfils the requirements of nature except in one respect: it has no means of adapting itself to different distances. We have learned (p. 57) that the nearer the object is to the refracting surface the further does the image on the opposite side of the surface recede from the surface. Hence if the retina is at such a distance from the cornea that distant objects are clearly focused on the layer of rods and cones, the conjugate focus for near objects must fall behind the retina. The rays of light in this case being intercepted by the retina before they are united in a focus, objects so seen must appear indistinct. In the physiological eye, adjustment to different distances is accomplished by the *crystalline lens,* which is capable, within certain limits, of undergoing increase of curvature to meet the requirements of correct focusing.

Lens-refraction. A lens is defined as a portion of transparent substance bounded by one plane and one curved surface, or by two curved surfaces, both centred on the same axis.

FIG. 32.



1 2 3 4 5 6

Lenses are classified according to the form of curvature: as *spherical, cylindrical, toric, elliptical, paraboloidal,* etc. Although lenses of the latter kinds of curvature have been made, the circle is the basis of curvature in practical lens construction, that is, artificial lenses are either *spherical, cylindrical,* or *toric. Spherical* lenses have the same curvature, and consequently the same refracting power in all meridians; *cylindrical* lenses have a circular curvature in the meridian at right angles to the axis of the cylinder, but no curvature in the direction of this axis; *toric* lenses lie between spherical and cylindrical lenses, that is, they have circular curvature in each of the two meridians indicated

[1] In reality the index of the cornea is considerably greater than that of the aqueous, and a divergent action is exerted by the refraction at the posterior corneal surface; but owing to the extreme thinness of the cornea, this refraction may be disregarded without material error, provided we assign to the cornea the lower index of the aqueous.

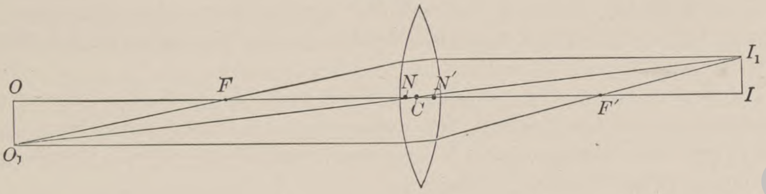
(the *principal meridians*), but the curvature is greater in one than in the other meridian.[1]

Lenses are classified also in respect of curvature, as: 1, *plano-convex*; 2, *bi-convex;* 3 and 4, *concavo-convex;* 5, *plano-concave;* and 6, *bi-concave* (Fig. 32).

Artificial lenses are made usually of glass and are surrounded by air; and since the refractive index of glass is greater than that of air, it is apparent that plano-convex and bi-convex lenses are collective in action, that plano-concave and bi-concave lenses are dispersive, and that concavo-convex lenses are collective (3, Fig. 32) or dispersive (4, Fig. 32) according as the convex or the concave refraction is greater.[2] In the former case the concavo-convex lens is called a *converging meniscus*, and in the latter a *diverging* meniscus. Menisci are called also *periscopic* lenses.

The formation of a real image by a collective lens is illustrated in Fig. 33. As in collective refraction by a single surface, a real image is formed when the first conjugate focal distance is greater than the principal focal distance. As the distance of the object increases the

FIG. 33.



conjugate focus moves nearer to the lens, and when the object is situated so far that the rays may be regarded as parallel, the image will be formed at the posterior focus, F'. When the rays are already convergent before entering the lens, the image will lie between the lens and the posterior principal focus. When the object is situated at the anterior focus F, the rays will be parallel after passing through the lens, and no image will be formed. When the object is within the anterior principal focus, the rays after passing through the lens will appear to come from a virtual focus—the image will be virtual.

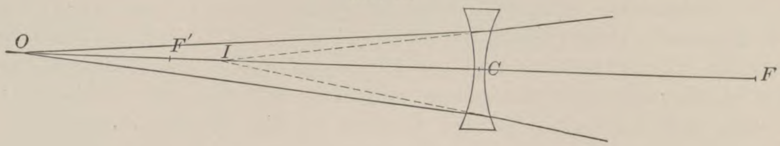
Since a dispersive lens increases the divergence of pencils, it is apparent that a real image can be formed after refraction by such a lens only when the rays have received, by previous or subsequent collective refraction, a convergence greater than the divergent action of the dispersive lens. The action of a dispersive lens is illustrated in Fig. 34. Rays proceeding from a point O, appear after refraction by the lens to come from I. When the distance $C\ O$ may be regarded as

1 Cylindrical and toric lenses will be considered more fully in another chapter

2 The exceptions noted in the case of refraction by single surfaces do not arise in lens-refraction; the result of the *two* refractions is always collective in the case of convex lenses and dispersive in the case of concave lenses.

infinite, I coincides with F', which is the posterior principal focus, since it is the virtual focus for rays which are parallel before refraction. The anterior principal focus is F (behind the lens), for rays which are directed toward this point before refraction are parallel after refraction.

FIG. 34.



Cardinal Points in Lens-refraction In refraction at a single surface rays passing through the centre of curvature undergo no refraction, and, as we have learned, the centre of curvature is on this account called the nodal point or the optical centre. But, since in a lens there are two refractions, the optical centre must be situated so that as regards any ray passing through it the refraction at the first surface must be exactly counteracted by that at the second surface. A ray, as $O_1 C I_1$ (Fig. 33), passing through the optical centre C, undergoes no change in direction, but a lateral displacement varying with the thickness of the lens.

There are two nodal points: the first one (N) is that point on the principal axis toward which the nodal rays are directed before refraction; and the second one (N') is the point from which these rays appear to proceed after refraction.

Any nodal ray, as $O_1 I_1$, is a secondary axis, and as the thickness of the lens becomes insignificant in comparison with the focal distances, the nodal ray approximates a straight line. In this case the two nodal points are merged in a single nodal point, coinciding with the optical centre. Hence when the thickness of the lens is disregarded (as in artificial lenses used in ophthalmology), the *cardinal points* are three in number: the nodal point (or the optical centre) and the two principal foci.

The principal focal distances are measured from the nodal point (which in this respect is also the principal point), and, as so measured, the two principal focal distances are equal, for, disregarding the thickness of the lens, the sum of the two refractions must be the same, whether one or the other of the two surfaces be exposed to incident rays.

Numeration of Lenses. The principal focal distance, or the *focal length* of a lens, measures its refractive power, the latter being inversely proportional to the focal length. The unit of measurement in ophthalmology is the *dioptre.* This represents the power of a lens having a focal length of one metre. A lens having a focal length of one-half metre is twice as strong as the unit-lens, and consequently has a power of two dioptres (2 D.); a lens having a focal length of two

metres has a power of 0.5 D., etc.[1] Collective lenses are denoted by the plus (+) sign, and dispersive ones by the minus (—) sign.

The Crystalline Lens. This is a bi-convex lens composed of fibrillar structure, increasing in density toward the centre or nucleus, the whole being enclosed in a transparent capsule and suspended by the suspensory ligament between the aqueous humor anteriorly and the vitreous body posteriorly. The refractive index of the crystalline lens as a whole—*the equivalent refractive index*—is about 1.438, while that of the aqueous and vitreous is about 1.337. Since the index of the bi-convex crystalline lens is greater than that of the surrounding media, this lens must exert a collective action upon the rays of light which pass through it after having undergone refraction at the cornea.

In the corneal refraction the anterior focus is about 23 mm. from the cornea, and since objects viewed by the eye are at a greater distance than this, it follows that the rays from any point of the object are already converging to a conjugate behind the cornea when they enter the crystalline lens. The convergence is increased by the collective action of the lens, so that the rays are brought to a focus sooner than they would be without the lens.

Compound Optical Systems. Several refracting surfaces centred in a common axis, and separated by intervals, as the various surfaces of the eye, constitute a compound optical system. It was first demonstrated by the mathematician Gauss that such a system is exactly analogous to a simple system, except that the anterior and posterior focal distances are measured not from a single point, but from *two principal points* separated by an interval. Similarly there are *two nodal* points, having the same significance as the nodal points of a thick lens (Fig. 33); but when, as in the eye, the final medium differs in index from the first, the nodal points do not coincide in position with the principal points, as they do in a lens; in this respect the eye is comparable to a single surface.

The Schematic Eye. The curvatures, indices, and positions of the refractive media of the human eye have been very accurately investigated. The following table presents the average values which have been determined for the normal adult eye.

CURVATURES.

Anterior surface of the cornea	7.8 mm.
Posterior surface of the cornea	6.0 "
Anterior surface of the lens during relaxation of the ciliary muscle	10.0 "
Posterior surface of the lens	6.0 "

Indices.	*Thickness.*
Cornea, 1.377	1.0 mm.
Aqueous, 1.337	2.6 "
Lens, 1.438	4.0 "
Vitreous, 1.337	

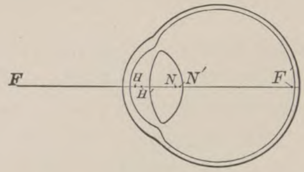
[1] In the old system of enumeration the *inch-lens* was taken as the unit of measurement. As lenses having a focal length of only one inch are seldom used in ophthalmology, the weaker lenses in common use had all to be expressed in fractions. Thus a lens of 1 D. (having a focal length of forty English inches) would be denoted by the fraction $^1/_{40}$; a lens of 2 D. would be expressed by the fraction $^1/_{20}$, etc.

By applying the formula of Gauss to these data the following are deduced as the cardinal points of the average normal eye:

First principal point (H)	1.8	mm. behind anterior surface of cornea.
Second principal point (H′)	2.1	" " " " "
First nodal point (N)	7.1	" " " " "
Second nodal point (N′)	7.4	" " " " "
Anterior principal focus (F)	14	" in front of the cornea
Posterior principal focus (F′)	23	" behind anterior surface of cornea.

A diagrammatic eye constructed in accordance with these measurements is called a schematic eye. (Fig. 35.)

Fig. 35.



The Reduced Eye. It will be noticed that the interval between the two principal and between the two nodal points is only 0.3 mm.; if we neglect this interval and merge the two principal and the two nodal points into a single principal and a single nodal point, the refractive effect of the eye is in all respects similar to that of a single surface of suitable curvature, the surface intersecting the axis where the principal points are merged, and the indices being those of the first (air) and final medium (vitreous), respectively. Such a substitution is called the *reduced eye*. The reduced eye is useful for the study of refraction, especially for experimental demonstration. The index of water is very nearly the same as that of the vitreous; hence we may make an artificial eye for demonstration by filling a suitable receptacle with water, the cornea being represented by a very thin spherical segment of glass, and the posterior face of the receptacle having a ground-glass face on which images are projected; or, for the study of the fundus, a painted representation of the retina and bloodvessels may be substituted. The curvature of the artificial cornea should be such that the focal distances are approximately equal to those of the normal eye. If water is the refractive medium, the radius of curvature should be about 5 mm.

Emmetropia. The posterior focal distance of the schematic eye is (approximately) 21 mm., and the second principal point, from which this distance is measured, lies about 2 mm. behind the anterior surface of the cornea; hence the posterior focus of the eye lies 23 mm. behind the corneal summit; that is, parallel rays will be brought to a focus at this distance from the cornea. If the retina coincides in position with this focus, the eye is adapted to receive a clear impression of a distant object. When this relation exists, the condition is called *emmetropia*. This is the ideal or normal state of refraction; but as this relation depends upon the curvature of the various surfaces as well as

upon the size of the eyeball, it is not to be expected that it uniformly occurs, even in healthy eyes. In fact, strictly speaking, emmetropia seldom exists, but it is only when the variation from the standard is capable of producing disturbance (visual or nervous) that the condition is to be regarded as abnormal; any deviation from emmetropia is called *ametropia*.

Aberration. We have, for the sake of simplicity, implied that all the rays of a refracted pencil meet the axis in a common point—the focus To fulfil this condition, there must be a suitable diminution of curvature, with increase of distance from the axis, for in spherical refraction the peripheral rays are proportionately too strongly deviated, so that they intersect the axis nearer the surface than do the central rays. This is called *spherical aberration*.

The refracting surfaces of the eye, while more nearly resembling ellipsoidal surfaces, differ at the axial portions only slightly from spherical surfaces, and are regarded as such in all calculations in the study of ocular refraction.

Function of the Iris. It is necessary, in order to procure a sharp image by spherical refraction, that all but the more central rays be excluded from the refracting media. This is accomplished in artificial systems by means of an opaque diaphragm having a circular opening of the desired size, through which the central rays are admitted to the refracting media. In the eye peripheral rays are excluded by the *iris*, the central rays being admitted through its central aperture—*the pupil*—which varies in size according to necessity. In bright illumination the pupil becomes very small, thus adding to the sharpness of the retinal image and preventing the dazzling of the retina which would occur from the excess of light. In feeble illumination the pupil dilates, so that, if possible, sufficient light may be afforded for the proper stimulation of the retina.

Chromatic Aberration. Besides spherical aberration, there is also chromatic or color aberration, which is due to the fact that the degree of deviation of light varies with the wave-length or color, violet being most and red least refracted. It may be experimentally demonstrated that color aberration occurs in refraction by the eye, but it is too slight to be noticeable in ordinary vision.

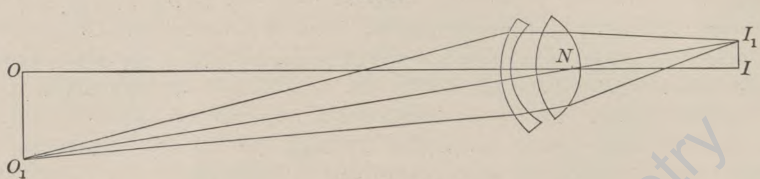
Increase of Aberration with Increase of Size of Object. Aberration is greater according as the secondary axes are the more removed from the principal axis. Hence it is evident that there is a limit not only to the size of aperture (the pupil), but also to the size of the object which will afford a clear image; *the object must always be small in comparison with the focal distances.* It is through the peculiar construction of the retina that we are enabled to see large objects with clearness. It is only the central portion of this organ, the *macula lutea*, lying near the principal axis, that is sufficiently sensitive to convey a well-defined impression to the brain. The macula lutea covers an oval area, about 2 mm. in the horizontal and 1 mm. in the vertical diameter, but not even all or the greater part of this area is concerned

in direct vision; the *fovea centralis*, upon which must fall the image of every object distinctly seen, is a minute depression near the centre of the macula. Thus only that part of the retinal image which is most sharply focused is utilized in *direct* vision. The less clearly formed portion of the image depicted upon the less sensitive periphery of the retina is, however, of great service in enlarging the field of *indirect* vision. Any object or part of an object lying in this field of indistinct vision, if it excites attention, is brought almost instantly by the muscular apparatus of the eye into the line of direct vision.

Function of the Choroidal and Retinal Pigment. The interior of a photographic camera is lined with black substance, by means of which light reflected from the plate is absorbed; otherwise by further reflections from the interior of the camera the plate would be affected by this unfocused light, and the image would be marred. In the eye this function is performed by the pigment of the choroid and retina.

Mental Projection and Rectification of the Retinal Image. It is apparent that the image as formed on the retina is an *inverted* image;

FIG. 36.



nevertheless, objects appear in their true relations as perceived by the visual sense. The rectification of the image is performed by the mind, possibly as the result of experience, in that the retinal image itself is not manifested to consciousness, but the external projection of this image—that is, *we do not see the image on the retina; we see the object.* Regarding the two nodal points as merged in a single point, the ray or straight line passing through the nodal point and connecting any point of an object with the corresponding point of the image marks the direction of the external point. (Fig. 36.) It is because this line, and only this line, represents, in normal vision, the true direction of an object that the mind has learned (through association of the visual sense with other senses) to project images along the nodal lines, and this even when, through artificial or pathological conditions, these lines do not indicate the true direction.

The estimation of the position—the distance—of an object is, likewise, not the result of any distinctive characteristics of the image, but is a mental product effected by the association of other senses and by the working of the two eyes in unison.

ACCOMMODATION.

We have alluded to the fact that the eye possesses the means of varying its focusing power according to the distance of the object viewed. This power of the eye is called *accommodation*. In the normal or emmetropic eye the image of a distant object (six metres or more) is focused on the retina, but the rays from a near object would come to a focus at some point behind the retina; the rays being intercepted by the retina before reaching their focus, the image as depicted upon the retina would be blurred. In order to afford a clear image the refracting power of the eye is increased by an increase of convexity of the crystalline lens, whereby the image is brought to a focus on the retina. The nearer the object of vision the greater must be the increase of curvature in order to adapt the eye for distinctness of image.

Mechanism of Accommodation. Accommodation takes place involuntarily (except as the result of special training) by reflex stimulation; the approach of an object before the eyes gives rise to an afferent impulse because of the mental desire for distinctness of the image. This afferent impulse conveyed by the optic nerve is transmitted (probably through the corpora quadrigemina) to the accommodation centre, this being the anterior portion of the nucleus of the third nerve. From this centre an efferent impulse is sent to the ciliary muscle, which, undergoing contraction, increases the curvature of the crystalline lens, and at the same time an impulse from the adjoining pupillary centre produces contraction of the sphincter pupillæ. Intimately associated with these two reactions is that of *convergence*, whereby the object is brought into the line of direct vision of each eye. So closely associated are accommodation and convergence that under normal conditions these two impulses are excited in unison; the accommodative impulse gives rise to convergence, and *vice versa*.

Helmholtz's Theory. The manner in which contraction of the ciliary muscle effects accommodation was first explained by Helmholtz. The crystalline lens is composed of fibrillar tissue. In early life the substance of these fibres is semifluid, so that the whole lens is of a gelatinous consistency. With increase of age the lens-substance hardens, first at the centre, forming a nucleus, and later the cortical portion also becomes firm, the lens being in old age a solid body incapable of undergoing change of shape.

The form of the soft lens is maintained by the capsule, an elastic membrane or sac in which the lens is enclosed. The capsule is attached peripherally to the ciliary muscle by means of the ciliary or suspensory ligament. (Fig. 37.)

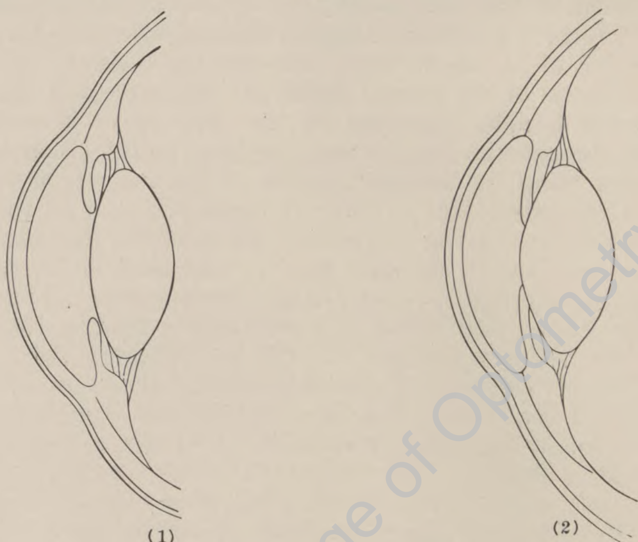
The *ciliary muscle* consists of two parts: the first or circular portion may be described as a ring-shaped muscle lining the inner surface of the sclera just behind the sclero-corneal junction; the second or longitudinal portion is composed of fibres which are united anteriorly with the circular portion, and which extend posteriorly to the equatorial

region of the eyeball, where they are inserted into the choroidal coat. In the normal eye the circular fibres predominate over the longitudinal ones in the proportion of about ten to one.

Assuming the sclero-corneal attachment to be the fixed point in the muscular action, it is apparent that contraction of the more powerful circular fibres must diminish the diameter of the ciliary ring, while contraction of the less potent meridianal portion of the muscle will, at most, produce a slight tension upon the choroid.

When the ciliary muscle is uncontracted, the anterior suspensory ligament is held tightly stretched, the posterior portion being much less so. (Fig. 37, 1.) The stretching of the anterior ligament causes a flattening of this surface of the lens; but when, by contraction of the muscle, the anterior ligament is relaxed, the anterior portion of the

FIG. 37.



(1) (2)

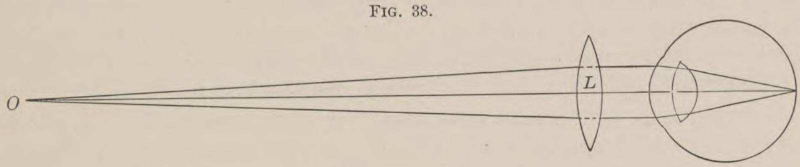
lens is allowed to bulge forward (Fig. 37, 2), so that the convexity of this surface is increased. In maximum relaxation of the ligament the form of the two lens-surfaces is practically the same, the radius of curvature being about 5.5 mm. in the young adult; for the posterior ligament also undergoes a slight relaxation, the radius of curvature of this surface being reduced from 6 to 5.5 mm.

Tscherning's Theory. Certain physiologists, most prominent of whom is Tscherning, believe that the theory advanced by Helmholtz does not afford the true explanation of accommodation. They believe that contraction of the ciliary muscle produces, by means of the longitudinal fibres, an increase of tension of the suspensory ligament, and that by this tension the curvature of the apices of the lens-surfaces is increased with a diminution of curvature at the peripheral portions.

Such a change is physically possible only in the event of the nucleus being firmly solidified while the cortex is fluid or gelatinous. This is not the condition of the human lens in childhood and early adult life —the period at which accommodation is most active; hence it would seem that this explanation is less plausible than that of Helmholtz.

Measurement of Accommodation. Accommodation is measured by the lens, which when placed in front of and as near as possible to the eye would have the same focusing power as the accommodation exercised. This is illustrated in Fig. 38. An object situated at O could be clearly seen by a normal eye with exercise of accommodation; without accommodation this eye would focus a distant object (parallel rays) on the retina. A convex lens (L) whose focal length is $L\,O$ would render rays from O parallel, so that the rays so rendered would be focused on the retina without accommodation. Hence the lens L has the same effect as the accommodation, and may be taken as the measure of the latter. If O is the nearest point for which an eye can accommodate, the lens L measures the accommodative power—the *amplitude of accommodation*—of the eye. If $L\,O$ is 0.25 metre in length, the accommodative power is 4 D., etc.

FIG. 38.



Variation of Accommodation with Age. Since accommodative power depends upon the ability of the crystalline lens to change its shape, it is apparent that this power must diminish as the lens becomes hardened with increase of age. At ten years of age there is normally an amplitude of about 14 D., that is, at this age an emmetropic eye can adapt itself for all objects distant not less than 71 mm. from the eye. Beginning at this early age there is a gradual diminution in accommodative power, and when the age of seventy or seventy-five years has been reached accommodation is no longer possible.

The following table (Donders) gives the accommodative power at intervals of five years:

Age	10	15	20	25	30	35	40	45	50	55	60	65	70	75
D.	14	12	10	8.5	7	5.5	4.5	3.5	2.5	1.75	1	0.75	0.25	00

Presbyopia. In ordinary near work, such as reading, the object of vision is usually at a distance of about $\frac{1}{3}$ of a metre (13 inches) from the eye, or even nearer in the case of very small print or other fine work. In order to adjust the eye for this distance, 3 D., or, at most, 3.5 D., of accommodation would be sufficient if one could use all his accommodative power continuously; but it is impossible to maintain the maximum of accommodative activity for more than a momentary period. It has been found that for long-continued near work only

about two-thirds of the total amplitude is available, and with advancing years a still smaller proportion can be utilized. If one attempts to engage in near work without this reserve accommodation, the eyes speedily tire, vision becomes blurred, and pain in the eyes, sometimes accompanied by headache, develops, so that the work must be abandoned. After a short period of rest work may again be resumed, with more or less prompt return of the aforementioned symptoms. If near work be persisted in under such circumstances, the symptoms will in time become very distressing, and to those already noted may be added extreme hypersensitiveness to light, and conjunctival congestion and inflammation, which frequently ensue.

In order that one may be able to use continuously 3 D. of accommodation he must have a total amplitude of 4.5 D. When from increase of age the crystalline lens has become so hardened that the amplitude falls below this amount (corresponding to vision at 22 cm., or 9 inches), the condition is called *presbyopia* (old sight). Reference to the table above given shows that the presbyopic state is reached when the fortieth year of life is passed; practically the condition is usually manifested between the ages of forty-three and forty-five years. It is often nearer the latter age when relief is sought, though the exact time varies according to the physical condition, to the character of work pursued, and especially to the refractive state of the eye.

The physiological condition of presbyopia should not be confounded with hyperopia, which may give rise to similar symptoms. A person having 3 D. of hyperopia will require (as we shall learn in Chapter III.) this amount of accommodation for distant vision; at thirty-five years of age the amplitude is 5.5 D., and if 3 D. of this must be used to focus parallel rays on the retina, only 2.5 D. will be available for the additional focusing required in near work. This amount being insufficient, near work becomes burdensome, *but this is not presbyopia;* the inconvenience arises not because the accommodation is weak, but because an abnormally high amount is required. With the aid of a convex lens correcting the hyperopia the symptoms disappear, to reappear, however, about the age of forty-five, when an additional convex lens will be required to take the place of the failing accommodation, that is, to overcome the presbyopia. On the other hand, a person who has 3 D. of myopia will never develop presbyopic symptoms, because he can focus rays coming from an object placed at the reading distance without any accommodation. Such a person will, however, become presbyopic (his accommodation will fall below 4.5 D.) at the usual age, and if he wears glasses correcting the myopia, he will have to remove these in order to read fine print.

Although presbyopia is a physiological condition, in that all eyes are subject to it, it would nevertheless entail most serious consequences among civilized races if it were not that artificial conditions of life have brought also artificial means of relief in the substitution of a glass lens for the loss of accommodative action of the crystalline lens of the eye.

The following table gives the probable strength of lens necessary in order to enable the presbyope to engage comfortably in near work:

Age	45	50	55	60	65	70	75
D.	1	2	2.75	3.25	3.5	3.5	3.5

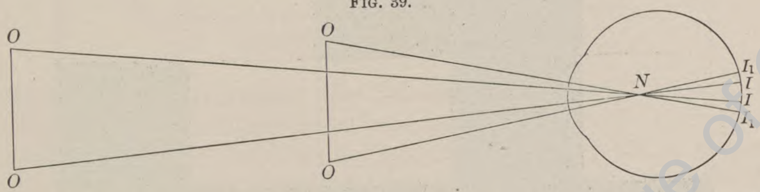
For reading, a glass of 3 D., or, at most, 3.5 D., is sufficient, even when the accommodative power is entirely lost; hence the rule usually given that 1 D. should be added for every five years is not applicable after fifty-five years. It is evident also that the strength of lens must vary according to the character of the work; it may be necessary in certain handicrafts to use a lens of 4 D., but a lens of this strength and even one of 3.5 D. would cause objects situated at a distance of one-half metre or more to be blurred, and for work which must be performed at such distance a lens of 2 D. would be required when all accommodative power is lost.

It is also apparent that in ametropia the presbyopic lens must be added to or subtracted from that correcting the ametropia according as this is hyperopia or myopia.

VISUAL ACUITY.

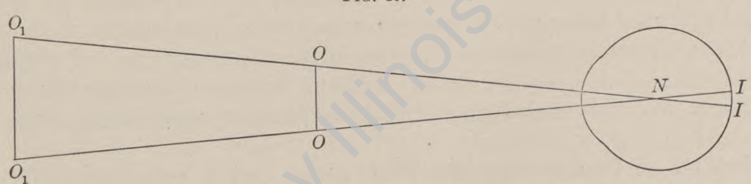
The size of the image as formed on the retina varies according to the distance of the object. Thus if $O\ O$ (Fig. 39) represents the

FIG. 39.



linear dimension of an object, the image of this dimension will be represented by $I\ I$, or $I_1\ I_1$, according to the situation of the object. Conversely, an object, $O_1\ O_1$, Fig. 40, will form on the retina an image of the same size as that of the object, $O\ O$.

FIG. 40.

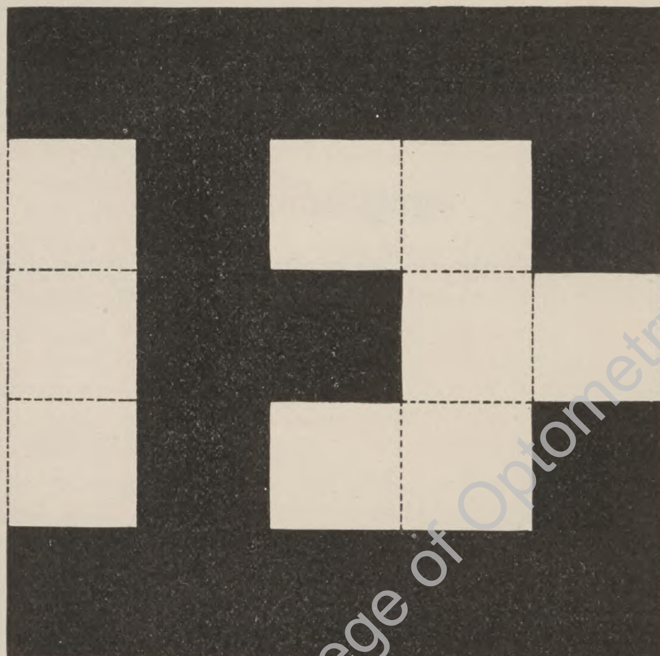


The Visual Angle. The angle $O\ N\ O$ (equal to $I\ N\ I$) is called the visual angle. The smallest angle which two points (as O and O) may subtend at the nodal point of the eye, while they are distinguished as separate, is called the *minimum* visual angle. The minimum visual angle measures the *visual acuteness* of the eye. It has

been found by experiment that under suitable illumination the smallest angle under which two white lines separated by a black interval can be distinguished as separate is for the normal human eye slightly less than *one minute.*

Test-letters for Measuring Visual Acuity. Making use of the foregoing experimental determination, Snellen constructed a series of test-letters so arranged that when placed at the proper distance each stroke of each letter would subtend an angle of one minute at the nodal point of the eye. This is illustrated in Fig. 41. When placed at a distance of 30 metres from the eye, each side of the square

FIG. 41.

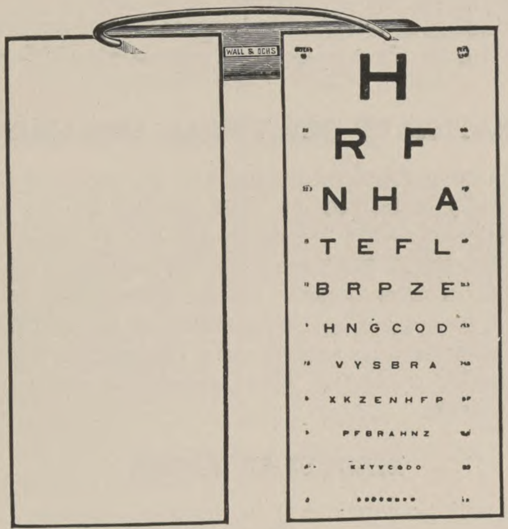


would subtend an angle of five minutes at the nodal point, and each stroke of the letter would subtend an angle of one minute. In testing visual acuteness, it is convenient to have letters of various sizes, the distance at which they subtend the one-minute angle being noted on the card. (Fig. 42.)

Method of Conducting the Test. The test is usually conducted with the letters placed at a distance of 6 metres. If at this distance an eye can distinguish those letters which subtend the one-minute angle, the visual acuity is normal. It is expressed by the equation $V = 6/6$ or $V = 1$. But if at this distance the eye can distinguish no smaller letters than those which subtend the one-minute angle at

12 metres, the visual acuity is only one-half as great as it should be; it is expressed by the equation V = 6/12. *In general*, the visual acuity is expressed by a fraction, the numerator of which is the distance at which the test is conducted, and the denominator is the distance at which the smallest distinguishable letters subtend the one-minute angle.

FIG. 42.



Thorington bracket with test letters.

Visual Acuity Exceeding the Standard. Partly because of the familiarity with the alphabetical characters and partly because the standard adopted by Snellen (one minute) is slightly larger than the minimum visual angle in young persons, it frequently happens that smaller letters can be read than those indicated for normal vision. Thus V = 6/4 or V = 6/3 may be recorded. In old persons vision exceeding 6/6 is not common, because of diminution of transparency of the media.

Estimation of the Refractive Condition and of the Accommodative Power by Means of Test-letters. Since an eye must have its maximal seeing power when the image is properly focused on the retina, we have in the test-letters a means of determining whether or not the emmetropic condition is present. If the visual power is increased by placing a convex lens before the eye, we know that without this lens the focus falls behind the retina (hyperopia); if the visual power is increased by a concave lens, the focus without the lens must fall in front of the retina, either from excess of curvature or of length of eyeball (myopia), or from undue action (spasm) of the accommodation; finally, if the maximal visual power is obtained with the aid of a cylindrical lens, the eye is, without the lens, adjusted to the object in the meridian of the axis of the cylinder, and hyperopic

or myopic (astigmatic) in the meridian at right angles to this, according as a convex or concave cylinder is required.

Having determined with the distant test-letters the refractive condition and the visual acuity of an eye, it is possible, by means of small letters constructed upon the same plan, to measure the accommodative power. Placing before the eye the lens which affords the condition of emmetropia, and noting the visual acuity, the same acuity should be obtained in near vision so long as the accommodative power is sufficient to adjust the eye for the distance at which the types are held.

DURATION OF THE VISUAL SENSATION.

The length of time required for light to produce stimulation of the retina is practically instantaneous; the shortest flash of light that can be produced experimentally is seen by the eye as perfectly as a much longer flash. Moreover, however brief the period of stimulation, the visual impression always persists for an appreciable interval (about one-eighth of a second) after withdrawal of the stimulus. Thus a series of rapid stimulations appears as a continuous stimulation—the spokes of a rapidly revolving wheel appear to cover every part of the area of the circle.

BINOCULAR VISION.

This subject will be considered in another chapter. It suffices to say here that in normal vision the muscular movements of the eyes are so associated as always to bring the image of an object (in direct vision) upon the fovea centralis of each eye. When this is accomplished, a single mental impression is received—slightly more intense and with better appreciation of form and perspective than is obtained from one eye acting alone.

CHAPTER III.

REFRACTIVE ERRORS IN GENERAL.

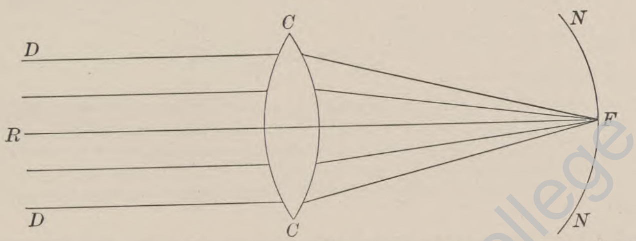
By ALEXANDER DUANE, M.D.

EMMETROPIA AND THE VARIETIES OF AMETROPIA.

When rays coming from a distant object—rays, that is, which are practically parallel to one another when they reach the eye—pass through the cornea and crystalline lens, they are brought together at the posterior focus of the eye, and form there a sharp inverted image of the object.

The retina may lie just at the posterior focus of the eye. The eye then is like a camera which is precisely focused for distant objects, and such objects, forming a sharp image upon the retina, will, if the eye is otherwise normal, be perceived distinctly. This condition, in which the eye naturally and without effort focuses parallel rays upon its retina, is called *emmetropia* (E.). (Fig. 43.)

Fig. 43.



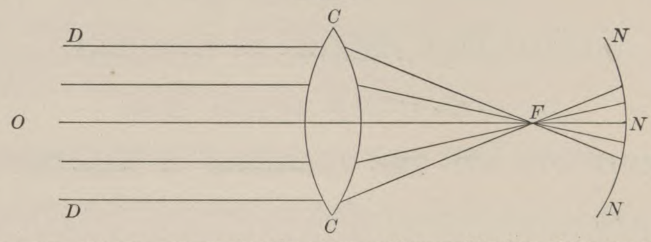
The emmetropic eye. *C C* is a lens representing the cornea and the crystalline lens collectively; *N N* is the retina lying at the principal focus, *F*, of *C C* (*F*= posterior focus of the eye). Rays coming from a very distant object, *R*, will be sensibly parallel to one another (thus taking the direction *D C*, *D C*) when they strike the eye, and will hence be sharply focused at *F*, so as to form a distinct inverted image of *R* upon the retina *N N*. The emmetropic eye is, therefore, like a camera which is accurately focused for distance.

If the point *F* on the fundus is illuminated so that it sends out rays in the reverse direction, *F C*, *F C*, these rays will emerge from the eye parallel to one another, taking thus the direction *C D*, *C D*, after passing back again through the crystalline lens and cornea.

Opposed to emmetropia is the condition known as *ametropia*, in which the retina is not at the posterior focus of the eye, and the eye consequently is not adjusted for parallel rays. An ametropic eye is like a camera out of focus, and cannot, except by accommodative effort or by the aid of a glass, form distinct images of distant objects upon its retina. Ametropia comprises the various *errors of refraction*, which are myopia, hyperopia and astigmatism.

Myopia (My.), or *nearsightedness*, is that condition in which the retina lies behind the posterior focus of the eye. The eye then is like a camera which is out of focus because its receiving plate is too far back of its lens. (Fig. 44.)

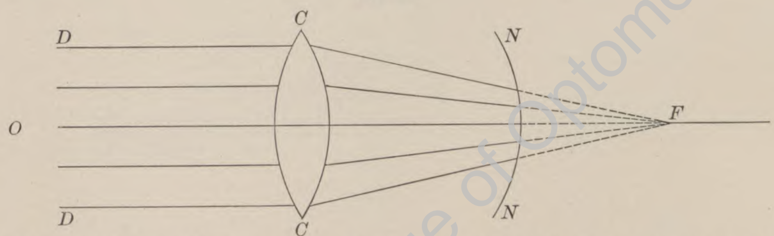
FIG. 44.



The myopic eye. *C C*, lens representing the cornea and crystalline lens of the eye collectively. *F*, the principal focus of *C C*, or of the eye, lying in front of the retina *N N*. Rays emanating from a distant object, *O*. and hence parallel to one another (taking the course *D C*, *D C*) when they reach the eye, are focused by *C C* at *F* in front of the retina. The eye is, therefore, not adjusted for *O*. The amount by which it is out of focus—*i. e.*, the distance between *F* and *N*—measures the amount of myopia.

Hyperopia, or *hypermetropia* (H.), also called *farsightedness* or *long-sightedness*, is that condition in which the retina lies in front of the posterior focus of the eye. The eye then is like a camera which is out of focus because its receiving plate is toö close to its lens. (Fig. 45.)

FIG. 45.



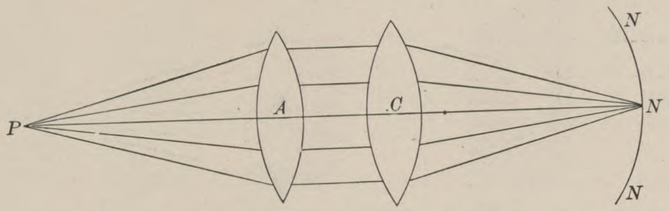
The hyperopic eye. *C C*, lens representing the cornea and crystalline lens collectively. *F*, the principal focus of *C C*, or of the eye, behind the retina *N N*. Rays emanating from a distant object, *O*, and hence parallel to one another before striking the eye (taking, therefore, the course *D C*, *D C*), will, after refraction through *C C*, be converged toward *F*. They will hence strike the retina before they come to a focus. The eye is, therefore, not adjusted for *O*. The amount by which it is out of focus—*i. e.*, the amount of its hyperopia—is measured by the distance between *N N* and *F*.

Astigmatism (As.) is that condition in which the several meridians of the eye differ from each other in refraction, so that each will focus parallel rays at a different point. Instead, then, of there being one principal focus for all meridians alike, as in myopia or hyperopia, there are a number of foci, one for each meridian, and these foci lie one behind the other. If the focus for any one meridian happens to lie upon the retina, that meridian will be emmetropic, while all the other meridians will be myopic or hyperopic, because their foci are in front of the retina or behind it. If the retina lies in front of all

the foci, all the meridians will be hyperopic, but some more than others ; and if the retina lies behind all the foci, all the meridians will be myopic, but some more than others. (Fig. 52.)

Accommodation. As we have seen in Chapter II., an emmetrope who wishes to focus for a near object does so by accommodating, that is, by increasing the convexity of his crystalline lens, and thus making his eye just that much the more refractive. In effect he adds to the lens another lens, *A*. (Fig. 46.) This lens *A* must give rays emanating from *P* such a direction that the eye *C* can focus them upon the retina *N N*. If the eye *C* is emmetropic or has been made so artificially—*i. e.*, if it is adjusted for parallel rays, *A* must make rays that diverge from *P* parallel to one another. But to do this, *A* must have a focal length = *A P*. Hence *the auxiliary lens which represents the accommodative effort that the eye makes in adjusting upon any near point must have a focal length equal to the distance of that near point from the eye.* This amounts to saying that when a patient adjusts for a point ten inches off, he practically adds to his eye by accommodative effort a 10″ (4 D.) lens; when he adjusts for half that distance, he adds a lens twice as strong, etc.

FIG 46.



Accommodation. The eye *C*, which is either naturally or artificially emmetropic, and therefore is adjusted for parallel rays, may be adjusted for the divergent rays coming from a near point, *P*, by exerting an amount of accommodation represented by the auxiliary lens *A*. This lens must have a focal length = *A P*, for then only will it make rays that diverge from *P* parallel—*i. e.*, give them such a direction that the lens *C* will be able to focus them upon the retina *N N*.

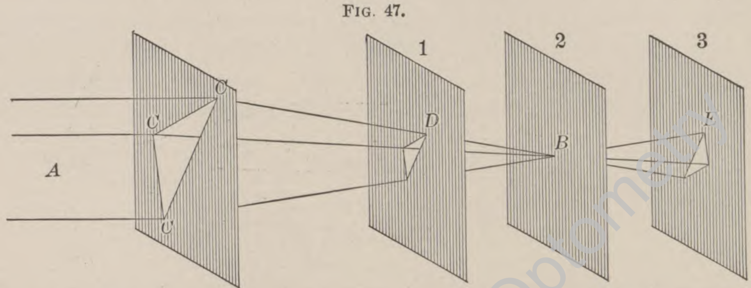
This conception of the accommodative process is very helpful in considering the correction of refractive errors. It is so far justified in that we may, if we wish, actually replace the accommodation by means of just such an auxiliary lens, and thus adjust the eye for any desired near point. Thus, suppose we paralyze the accommodation completely in an emmetrope with atropine, so that his eye unaided can see distinctly at distance only. Then, by placing a 5 D. (8″) lens before his eye, we at once adjust his sight for a point 8″ from him, and enable him to read at that distance, just as if he were using an equivalent amount of accommodation.

Vision in Ametropia. Diffusion Images. An uncorrected ametrope always sees in diffusion images. To understand what this means, we have only to consider what happens when the rays emanating from a distant point, *A* (Fig. 47), strike an eye which has no great amount of astigmatism. These rays, impinging upon the cor-

nea, are converged, then pass through the pupil and, striking the lens, are converged still more, so as finally to unite pretty sharply in a point, B, situated at the posterior focus of the eye. They thus form a conical or pyramidal bundle whose base is formed by the pupil C, and whose apex by the point B. The cross-section of this bundle will have the same shape as the pupil, being, therefore, usually circular, but, in cases of irregular pupil, being oval or any other shape, D.

In emmetropia the retina is situated at the posterior focus of the eye, and will intersect the bundle $C\,B$ at B, where all the rays of the bundle unite in a single point. Consequently, the retinal image of the point A, which image is formed by the union of the rays coming from A and forming the bundle $C\,B$, is a single sharply defined point of light. A distant object, being made up of a series of points, such as A, will then form upon the retina a series of sharply defined points like B, each one of which resembles its original in arrangement and distinctness. Hence the retinal image will be a true and clear representative of the external object.

FIG. 47.



Diffusion images. The rays emanating from a distant point, A, pass through the triangular pupil $C\,C\,C$, and are converged to form a pyramidal bundle of rays, $C\,B$, $C\,B$, $C\,B$, uniting in a sharp point at B. If the retina is at 2 (emmetropia), the image of the point A is the point B. If the retina is at 1 (hyperopia), the image of A is the triangle D, which is larger and more hazy in proportion as D is in front of B. If the retina is at 3 (myopia), the image of A is the inverted triangle E. D and E are diffusion images.

It will be otherwise in ametropia. Here the retina is either in front of B (in hyperopia) or behind it (in myopia). In hyperopia the retina, intercepting the rays before they come together, will have formed upon it, as the representative of the point A, a figure, D, of the same shape as the pupil. It is evident that the further off D is from B—*i. e.*, the greater the hyperopia—the larger D will be, and therefore the fainter, too, since all the light that in emmetropia is concentrated in the one point B, is now scattered over a comparatively large area.

Similarly in myopia the retina, being behind B, will have formed upon it a figure, E, of the same shape as the pupil inverted, and bigger and fainter in proportion to the degree of the myopia.

The faint, enlarged images D and E, formed on the retina in

ametropia, and representing a single point of light, are called diffusion images.[1]

In astigmatism the shape of the diffusion images will depend upon the amount of ametropia in the different meridians and the shape of the diffusion images upon the direction in which the least ametropic meridian lies. The special varieties that occur will be discussed later on.

In ametropia the retinal image of the distant object will be made up of a series of overlapping diffusion images, which will more or less confuse one another. Hence such an image will be blurred, and the more so the greater the size of the diffusion images.

The size of the diffusion images is dependent not only on the degree of the ametropia, but also on the *size of the pupil.* For it is evident that the smaller the latter—*i. e.*, the smaller the base of the cone *C B*—the smaller will be the sections *D* and *E*. This shows us why in ametropes, and likewise in presbyopes, who for objects within their near point also see in diffusion images, vision with contracted pupils is much sharper than when the pupils are dilated. Indeed, an ametrope of even high degree, provided his visual perception is intact, will see nearly as well as an emmetrope, if only his pupil is contracted *ad maximum* or is made artificially small by the use of a pinhole (stenopæic aperture).[2] This fact is utilized when in testing a patient we wish to ascertain whether he sees poorly because we have not yet given him the proper glass, or because, owing to some imperfection of the media, retina, or nerve, the seeing power itself is impaired. In the former case the pinhole will improve the sight, in the latter case it will not.[3]

It is for this reason, also, that myopes try to improve their sight by squeezing their lids together. This in effect narrows their naturally wide pupils and gives them smaller diffusion images. Hyperopes, who have naturally small pupils, and who besides can get around their difficulty by using their accommodation, generally do not need to employ this device.

For this reason, also, the vision in ametropia is usually worse in a dim light, when the pupils dilate. And many ametropes and presbyopes secure good vision by using a strong light which falls directly upon the eyes, and so contracts the pupils to pinpoints.

Resuming, we may say: *In ametropia the retinal image of a distant point will be a diffusion image whose shape will be the shape of the pupil, and whose size will be directly proportional to the degree of ametropia on the one hand and to the size of the pupil on the other.*

The resulting blurring of sight will be directly proportional to the size of the diffusion images.

1 Usually called *diffusion circles;* but, as pointed out, while generally circular, they may have any shape, depending upon the shape of the pupil.

2 Some of the first spectacles were nothing but pinhole apertures in metal plates. With these a myope could see distinctly at a distance, and a presbyope could read.

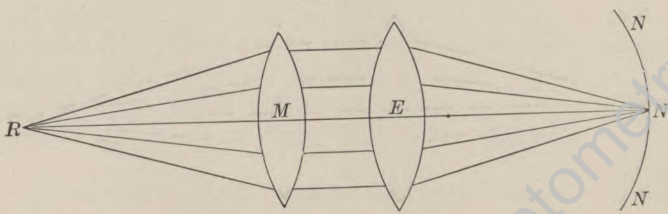
3 Theoretically; but, as a matter of fact, the pinhole cuts off so much light that the test is not a very serviceable one.

Correction of Ametropia. To obviate this blurring and enable the ametrope to see perfectly at a distance, we must in some way abolish his ametropia. This is effected sometimes by the accommodation, sometimes by the use of glasses. However effected, the correction practically converts the ametrope into an emmetrope, and he should see like one both for distance and near.

Myopia. Far Point in Myopia. The myope, as we have just remarked, sees a distant object in diffusion images. Such objects, therefore, appear blurred to him, and the more so the higher his myopia.

But while the myope sees poorly for distance, he sees well for near. For if the eye is such that parallel rays entering it focus at F in front of the retina (Fig. 44), rays that diverge from some comparatively near point, R, will focus back of F, and, if F is sufficiently near the eye, will focus right upon N N. The eye, in fact, is like a camera which is adjusted not for distance, but for the nearer object R. It is practically in the same condition as the accommodating eye (Fig. 46), and, like the latter, may be regarded as equivalent to an emmetropic eye to which a convex lens has been added. (Fig. 48.) Such a con-

FIG. 48.



The myopic eye considered as an emmetropic eye, with a convex lens added. The myopic eye may be regarded as an emmetropic eye, E, with a convex lens, M, representing the myopia added. Such a lens will collect rays coming from its focus, R, and render them parallel, when E, the emmetropic portion of the eye, will focus them upon the retina, N N. The eye is thus, by its excess of refraction (represented by the lens M), naturally adjusted for a point, R, which is, therefore, its far point. The focal length of $M = M$ R—*i. e.*, the amount of extra refraction produced by the myopia—is equivalent to a convex lens whose focal length equals the distance of the far point from the eye.

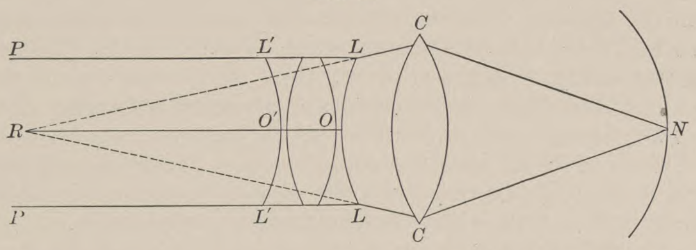
ception of myopia, while not absolutely accurate, is yet essentially so, and is so far justified in that we can imitate quite precisely the conditions of a myopic eye by actually placing an appropriate convex glass before an emmetropic eye. Thus if an emmetrope wishes to have an idea of how a myope of 8 D. sees, let him place an 8 D. convex glass before his own eye. Distant objects will at once appear altogether blurred and indistinguishable, being seen simply in outline or only as a uniform blotch of black and white; while objects just within 5″ will appear not only distinct, but also magnified, and furthermore will be seen without accommodative strain.

In fact, so far as vision is concerned, it makes little difference whether one is continuously using 2 D. of his accommodation (Fig. 46), or has a + 2 D. glass before his eye and uses no accommodation, or

is myopic 2 D. (Fig. 48.) In each instance he will be adjusted for a distance of 20″; at this distance he will see clearly, and beyond it will see indistinctly, because he sees in diffusion images.

This surplus of refractive power, or extra lens, M (Fig. 48), that a myope possesses over and above an emmetrope, is the measure of his myopia. It also gives us directly the point for which his eye is adjusted without the exercise of any effort of accommodation—*i. e.*, gives us his *far point*. For if R is the far point, then M and E together will focus upon N N rays that emanate from R. But to do this, M must make these rays parallel, for then E, the emmetropic quota of the eye which is adjusted for parallel rays, will focus them properly. If, however, M is a lens that renders rays coming from R parallel, R must be its principal (anterior) focus. That is, *the myopic eye may be represented as equivalent to an emmetropic eye to which has been added a convex glass having such a strength that its principal focus will lie precisely at the far point of the eye.*

FIG. 49.



Course of emergent rays in myopia. Correction of myopia. R, the far point of the myopic eye. Rays $R\ C$, $R\ C$, diverging from R will be focused by $C\ C$ (representing the cornea and crystalline lens collectively) upon the retina N. So, also, if N is illuminated and sends out rays, $N\ C$, $N\ C$, these after passing back through $C\ C$, and leaving the eye, will be converged so as to meet at R (principle of conjugate foci) and will form there a real inverted image of N. L, the concave glass correcting the myopia, placed at the normal distance (one-half inch) in front of the eye. L will give parallel rays, $P\ L$, $P\ L$, a divergence as if they came from R—*i. e.*, will make them take the direction $L\ C$, $L\ C$. $C\ C$ can then focus these rays upon the retina N just as if they had started from R in the first place. R is hence the principal focus of L, whose focal length, therefore, is $O\ R$. If L is shifted half an inch further out (to L'), its focus must still be at R, and its focal length will have to be $O'\ R$ —*i. e.*, it will have to be a stronger lens than L in order to give parallel rays the proper direction.

Correction of Myopia. The foregoing conception enables us at once to deduce the way to correct myopia. If myopia consists in an excess of refractive power, such as is represented by a convex lens, M, it will be corrected by a glass that will perfectly neutralize M—*i. e.*, by a concave glass of the same strength or focal length. This can be seen even more readily in Fig. 49. Here R represents the far point of the eye, and L the lens correcting the myopia. Since L corrects the myopia, or, in other words, renders the eye emmetropic, it must adjust the eye for parallel rays. As, however, the eye is naturally adjusted for rays that are already divergent, and, in fact, for rays, $R\ C$, $R\ C$, diverging from R, the correcting glass, L, must be such as to make parallel rays diverge as if they came from R. Such

a glass must be a concave lens, and its focus must be at R. We see, therefore, that *the glass which corrects the myopia of an eye must be a concave lens whose focus is at the far point of that eye.*

Myopia is usually measured by the strength of the glass that corrects it. Thus we speak of a myopia of 4 D., meaning an eye whose correcting lens is a — 4 D., and whose far point consequently is about 10″ in front of the eye.[1]

The efficiency of a glass in correcting myopia will vary somewhat according to the distance of the glass from the eye. For, no matter where the correcting glass L is situated, it must have its focus at R, the far point of the eye, in order to give parallel rays the proper direction for the eye to focus them. If, then, L is pushed away from the eye, say to L', its focal length, instead of being $R\ L$, it will be the shorter distance $R\ L'$—*i. e.*, it will have to be a lens of shorter focus, that is, of greater power, in order to do the same work. For instance, if a given myopia is corrected by a glass of — 10 D. (= 4″ focal length) placed ½″ from the cornea, it will need a glass of 3½″ focal length (= 11.5 D.) to correct the myopia when the glass is placed 1″ from the cornea. That is, what would be a proper correcting glass if placed ½″ from the eye, has become 1.50 D. too weak when shoved ½″ further away. We see, then, that *the strength of a concave glass—i. e., its ability to correct myopia—is lessened if the glass is carried away from the eye, and is increased if the glass is brought nearer the eye.* This is true whether the concave glass is used for distance or for near. As will be seen from the above example, the difference produced in this way with strong glasses is considerable.

It is for this reason that we see persons whose glasses do not fully correct their myopia pushing the glasses close in against their eyelids in order to see distinctly at a distance. They thus in effect increase the effect of their glasses.

Accommodation in Myopia. Near Point. The myope can, without using any accommodation at all, see distinctly an object situated comparatively near him, namely, at his far point. He can still, therefore, see quite near objects sharply, even if his accommodation is paralyzed with atropine. If, however, he uses his accommodation, he can focus down upon points still nearer. When he uses all his accommodation his eye is adjusted for his *near point*. Obviously this will be nearer to his eye than is the near point of an emmetrope having an equal amount of accommodation. Thus an emmetrope having 4 D. of accommodation can, by using the utmost accommodative effort, see an object situated 10″ from his eye. A myope of 4 D. will be able to see an object at this distance without using any accommodation at all; and if he does use 4 D. of accommodation in addition, he will be able to see an object at 5″ from the eye. For, as compared with the emmetrope, who in using all his

[1] This statement is not quite accurate if, as should properly be done, the distance of the far point is reckoned, not from the point where the glass is usually placed, but from the nodal point of the eye, which is an inch further back.

accommodation has in effect placed a +4 D. glass before his eye, he is like a person who has a +4 D. glass (representing the surplus of refraction due to his myopia) combined with another +4 D. glass (representing his accommodation)—*i. e.*, he is in effect an emmetrope with a +8 D. glass before his eye, or an emmetrope who is using 8 D. of accommodation.

A myope's *range of distinct vision* is obviously very limited, even when the nearsightedness is of low degree. Thus a myope of 2 D. who has 6 D. of accommodation, and has, therefore, a far point at 20″ and a near point at 5″, can see distinctly through a range of only 15″. The higher the myopia the more this range diminishes, and in fairly high degrees it becomes practically *nil*. Thus a myope of 10 D. with an accommodation of 6 D. would have a range of only 1.5″ (from his far point at 4″ down to his near point at 2.5″).

Some compensation for this limited range is found in the fact that a myope in doing near work is partly or wholly *independent of his accommodation*. Thus a myope of 4 D., as he sees distinctly at a distance of 10″ without using any accommodation, will never need to have a glass for reading, no matter how old he becomes. A myope of even 2 D. will not need to use glasses for reading nearly so soon as an emmetrope will. For, while the latter usually has to get glasses when his accommodation is reduced to 4 D., or when he is about forty-three years old, the myope of 2 D. will not have to get a glass until his accommodation is reduced to about 1.5 D.—*i. e.*, at the age of fifty-five years. For the same reason we can use atropine or homatropine in myopes with considerable freedom, as we can assure them that the instillation, even though it does abolish the accommodation, will cause them little or no interference with near work.

As myopes, and particularly myopes of fairly high degree, need to use their accommodation so little, they have not the same facility in this regard as emmetropes and hypermetropes. When, however, we correct a myope we convert him suddenly into an emmetrope, and he has to accommodate like one. Naturally this sudden assumption of an almost disused faculty is not easy for many, and, indeed, most myopes find difficulty at first in using their glasses for near work. The older the patient, the more pronounced this difficulty is. It is astonishing, however, with what ease most myopes reacquire this ability to use their accommodation, many doing so at once, and nearly all doing so in a very short time. When, however, the nearsight exceeds 12 D., we frequently find that the myope, especially if he is of adult age and has not used suitable correcting glasses, cannot, even after persistent effort, learn to use as strong a glass for near as for distance. In these cases the accommodative faculty is actually lacking, and this is due, as anatomical researches have shown, to atrophy of the ciliary muscle.

In myopia of low degree the accommodation is usually quite active, and may often, indeed, act *excessively*, producing an apparent exaggeration of the nearsight. The accommodative effort, in other words,

will cause an excessive bulging of the crystalline lens, and thus add just so much to the refractive power of the eye, already too great. This factitious or accommodative myopia, produced by spasm of accommodation, will be considered later. (See under "Varieties of Myopia.")

One effect of the disuse of accommodation in myopia is to produce an *accommodative convergence-insufficiency*, or exophoria, which may develop into a regular divergent squint, which at first is periodic (marked only for near), afterward constant. An exophoria, and in its incipiency[1] a divergent squint, having this origin may be corrected by the use of concave glasses, which compel the patient to use his accommodation, and hence also to converge.

Varieties and Causation of Myopia. We have regarded the myopic eye as an emmetropic eye with a surplus of refractive power added to it. This is true of many cases. That is, in these cases the eye is of normal length, but the cornea or lens has its refractive power increased, so that the two together focus too strongly or bring the rays together too soon. This overplus of refractive power may be due to excessive bulging of the surfaces of the cornea or lens (curvature myopia), or to changes in the density of the lens, cornea, or aqueous, altering their index of refraction (index myopia). In most cases of myopia, however, the cornea and lens are of nearly normal curvature and density, the eye being myopic simply because it is too long, so that the rays, although normally focused, come together in front of the retina (axial myopia).

A transient *curvature myopia* is produced in what we call spasm of accommodation; when the patient, by excessive use of his accommodation, temporarily increases the curvature of his crystalline lens above the proper amount. This *accommodative myopia* may happen (1) as the result of excessive near work; or (2) from the effort of trying to see by a poor light; or (3) of trying to see when the sight is obscured by an opacity of the cornea or lens. It may also (4) result from the effort to see more distinctly in astigmatism, and (5) it frequently is produced by the effort of accommodation expended in overcoming a concave glass. An emmetrope or a hypermetrope with good accommodation can see as well with a —1 D. glass as without it, because he almost immediately contracts his ciliary muscle, bulges out his crystalline lens, and thus gives his eye an additional refractive power of 1 D., which, acting like a convex lens of that strength, neutralizes the concave glass. By a similar process a man who is actually myopic 1 D. will see as well with a —2 D. as with a —1 D. glass.

The spasmodic contraction of the ciliary muscle by which a man may thus apparently increase his myopia if he is really myopic, or simulate a myopia when he is actually emmetropic or hyperopic, is called *spasm of accommodation*. It may be transient or last for a considerable period—years even—according as the causes which

[1] And even if of several years' standing.

produce it are transitory or permanent. Homatropine, or, in the case of a persistent spasm, atropine, repeatedly instilled will abolish the spasmodic contraction of the muscle and with it the factitious myopia. This means of diagnosis should always be employed when there is a suspicion of spasm of accommodation, and, as experience shows us that in people below forty-five years of age a spasm of this sort very frequently exists, it is important to use homatropine or atropine whenever we can in our examination of the refraction. Otherwise we shall often estimate the myopia too high or the hyperopia too low. (See remarks upon "The Use of Cycloplegics," later on.)

Permanent curvature myopia is usually dependent upon structural changes in the cornea or lens, due to disease. Examples are the myopia of conical cornea and that associated with many opacities of the cornea, and the myopia produced by a crystalline lens which is dislocated, and, being thus freed from the tension of its suspensory ligament, bulges out because of its own elasticity. Permanent curvature myopia is associated almost always with astigmatism, often of the irregular variety.

An example of *index myopia* is that often occurring in the development of cataract, when the lens, owing to alterations in its density, and especially to sclerosis of its nucleus, becomes more refractive. This myopia, which may amount to several dioptres, is often also associated with astigmatism, as the increase in density does not take place in all parts of the lens to the same degree.

The transient myopia often developing in iritis[1] is held by some to be an example of index myopia, the increase in refractive power being attributed to increased density of the aqueous. It is doubtful, however, whether this explanation is valid.

Axial myopia is by far the most common variety. It is due to the gradual elongation of the eye which occurs in childhood and in youth, and which causes a gradual recession of the retina. This process occurring normally in all eyes, occurs excessively in myopes, and, as it goes on, tends to make them more and more nearsighted. Every millimetre of such recession corresponds to an increase of about 3 D. in the myopia.

The elongation in myopia affects almost exclusively the back part of the eye, which loses its globular form and bulges out in the form of an egg. The end of this egg-shaped figure lies about at the yellow spot, and the parts in the vicinity of the latter consequently suffer the most from the stretching produced by the elongation of the eye. The parts in front of the lens, on the contrary, suffer little change, the cornea retaining its curvature, and the anterior chamber its normal depth.

The reason for this excessive elongation of the back part of the eye—the reason, that is, for the development and progress of myopia—has not been satisfactorily determined. There must be some cause,

[1] Amounting sometimes to 2 D. or more.

either external force or internal pressure, acting to distend the back of the eyeball. This cause has been variously considered to be the pressure of the external muscles, especially the obliques, undue accommodative action, the traction of the optic nerve, etc.

Whatever the cause, it is held usually to be one that is particularly active when the *eyes are used for near work.* Statistics are forthcoming in abundance which seem to show that myopia increases *pari passu* with the amount and complexity of the work to which the eyes are subjected. Thus it has been made quite certain that myopia is of lower degree and also less prevalent in the lower schools than it is in the higher schools and in the colleges; and the inference has been drawn that the myopia results in the first instance from the moderate application of the eyes in the lower schools, and is then enhanced by the further and greater strain imposed upon the eyes by the more complex work of the higher schools. This inference, although perhaps, in part, correct, is not really warranted by the statistics, for myopia, being a progressive affection, and increasing naturally with age, would of necessity be more frequent and of higher grade in the older pupils, whether they used their eyes for near work or not. Moreover, it is quite frequently the case that myopia, especially myopia of high degree and rapid progress, develops in those who do not use their eyes for near work at all. Thus it is fairly common in peasants who lead an out-of-door life and who cannot read nor write. The influence, then, of near work in producing myopia, although undoubtedly marked, has probably been considerably overrated.

In any case, the use of the eyes for near work is not the sole cause of myopia. It is at most the *exciting cause.* For of two children of the same age, and both subjected to the same kind of work in the same school, one will become nearsighted, and the other will remain hyperopic or become simply emmetropic. There must be some *predisposing cause* acting in the former case to produce the myopia. As such predisposing causes have been alleged the shape of the orbit (a low, broad orbit being supposed to favor the development of myopia), abnormal insertion of the external muscles, especially of the obliques, insufficiency of the interni, etc. A more important cause probably than any of these is a natural distensibility of the back of the eye, allowing it to recede under the influence of even a normal pressure. Whatever the predisposing causes are, they seem to be *inherited,* for myopia, and particularly the rapidly progressive form of it, tends to run in families. Consanguinity in the parents also seems to have some effect in producing it.

Complications of Myopia. Myopia is associated quite frequently with complications of the fundus. The chief of these are the so-called crescent or conus; rarefaction and other degenerative changes of the choroid, grouped under the name of sclero-choroiditis posterior; actual central choroiditis; hemorrhages in the choroid or retina; the formation of a black spot, probably due to pigment proliferation in the macula lutea; and detachment of the retina.

Of the *conus*, three varieties may be distinguished :

First, there is the congenital form, skirting the lower border of the papilla. This variety, which is attributed to an arrest of development (non-closure of the fœtal fissure), is usually associated with a moderate degree of myopia and astigmatism, although it often also occurs in hyperopic eyes. The myopia found with a congenital conus may be progressive, although in many cases it remains stationary through life, and the conus inferior as such has no special significance in indicating an advance of the myopia.

The second form of conus occurs as a moderately large, sharply defined white crescent, skirting the temporal, or, more rarely, the nasal, side of the disk. It may be combined with the inferior conus, or it may encroach on the upper border of the disk. This form is the regular concomitant of myopia of moderate degree, that is, up to 10 D. Yet it is absent in not a few cases of myopia, even in those of considerable amount, and frequently is found with emmetropia or with hyperopia. In itself, if unassociated with rarefaction of the adjacent choroid, it does not argue an active progression of the myopia, and is not to be regarded as pathological.

The third form of conus is the large triangular or irregularly oval white patch of atrophy with ill-defined borders, or with a series of concentric borders (terraced conus). This may occupy the temporal side of the papilla or spread so as to envelop the latter on all sides (annular conus). It is found regularly with myopia of 10 D. or more, sometimes also with myopia of less amount. It always indicates progression of the myopia, and is to be regarded as one of the manifestations of a sclero-choroiditis posterior, other evidences of which are almost invariably present. It is, therefore, a pathological phenomenon.

Sclero-choroiditis posterior, under which term may be included all forms of rarefaction and atrophy of the choroid, with or without the deposition of pigment, occurs regularly with myopia of more than 10 D., and sometimes also with myopia of only 5 or 6 D. Whenever present, it indicates advance of the myopia, and shows that we are dealing with an abnormally distensible or, at all events, an abnormally distending eye. Very rarely indeed a myopia of more than 10 D. is found without conus and without evidences of rarefaction of the choroid.

The other fundus changes—viz., hemorrhages, degenerative and inflammatory alterations in the yellow spot, and detachment of the retina—occur with moderate frequency in myopia. Statistics seem to show that, contrary to the usual opinion, these accidents are not much more common in the very high degrees of myopia than in myopia of 5 or 6 D. But inferences drawn from statistics are not entirely trustworthy. These accidents are more likely to occur after the ordinary changes of myopia (sclero-choroiditis posterior, etc.) have lasted a long time.

Progress of Myopia. Myopia is rarely congenital. Some of the cases associated with an inferior conus, no doubt, date from birth,

and some other congenital cases are probably due to disease of the eye occuring in utero. But in the overwhelming majority of cases myopia is an acquired affection which develops between the ages of five and twenty. Three classes of cases may be distinguished.

In the *first class* of cases the myopia never exceeds 2 D. Such a myopia may develop in late childhood or in youth, in which case its advance, if not arrested sooner, ceases at the age of twenty-one or twenty-two, when the patient attains full growth. A myopia of this sort may also start in adult life, and is then generally the result of excessive use of the eyes for near work. This low myopia is usually associated with astigmatism. It is probable that this association is not fortuitous, but that the astigmatism is the cause of the myopia ; that is, a patient starting with hyperopic astigmatism, converts this in his efforts to see distinctly first into a mixed and then into a myopic astigmatism. Myopia of this amount is not usually associated with a conus.

In the *second class of cases* the maximum pitch to which the myopia attains is about to 2 to 10 D. Such myopia develops in childhood, especially during the school age, and tends to increase up to the age of twenty-one or twenty-two, when, with rare exceptions, it comes to a stop. Its advance appears to be directly proportional to the demands made upon the eyes in school-work. Hence myopia of this kind is often called "school myopia." Yet, as before remarked, there is probably no actual intimate relation in most cases between school-work and the progress of myopia. Excessive near work may initiate a myopia, but is not probably the main factor in causing its advance after it has started.

Myopia of this kind is often accompanied with astigmatism. It is generally associated with a simple temporal or nasal conus, and not with the progressive (large, terraced, or annular) conus, nor with true sclero-choroiditis posterior.

In the *third class of cases* the myopia begins in early childhood, increases rapidly during the growing period (often reaching 10 or 12 D. at the age of ten), and so far from coming to a stand-still at twenty-one, keeps on increasing in adult life, so as ultimately to attain 15 to 20, or even 25 to 30 D. This kind of myopia bespeaks an unusually yielding and distensible eye. It is almost invariably associated with a large conus and marked sclero-choroiditis posterior, which may develop long before the myopia has reached a high grade.

In contradistinction to the other kind, this form of myopia is denoted as *progressive* or *pernicious*.

This form is distinctly pathological. It does not develop particularly as the result of excessive near work, and, indeed, occurs comparatively often in those who use their eyes but little. It occurs more often in the foreign born than in native Americans, and more often in dispensary patients than in the well-to-do. It is, in fact, a vice of development.

The elongation of the eye in many, and probably in most cases of myopia, takes place *discontinuously*, so that the nearsightedness will remain at the same point for perhaps several years, and then suddenly make an advance. This is shown in many cases by the presence of a terraced crescent adjoining the papilla, each terrace representing a period of renewed progress.

It should be noted that not all cases of progressive myopia are due to elongation of the back of the eyeball. The advance may be due to rapid increase in the corneal curvature, as in true lenticonus, and also in certain other cases not strictly classifiable under this head. It should also be remembered that mixed cases of curvature and axial myopia, due to changes in the curvature in the cornea and lens combined with elongation of the eyeball, are not uncommon.

Vision in Myopes. Myopes have very hazy sight for distance. The myope of 2 D. rarely has more than 2/100 vision; one with 4 D. cannot count fingers across the room; and in the higher degrees of nearsight the blurring is still more pronounced. This blurring is enhanced by the dilatation of the pupils which is usually present in such cases, and which acts by enlarging the diffusion images.

For *near points* myopes have very good vision—in fact, sharper vision than emmetropes, since near objects appear to them larger than they do to emmetropes. Moreover, as myopes do not have to use their accommodation, even when the object is quite near, they see such an object without accommodative strain. As an offset to this, it often happens that they have to hold an object so close to see it distinctly that they cannot keep both eyes converged upon it, and, hence, get into the way of looking with only one eye at a time, allowing the other eye to diverge. They thus have only monocular vision.

With glasses, the vision in myopia of low and medium degrees is generally nearly or quite normal. In myopia of over 12 D. the vision is seldom better than 20/40, and in myopia of 20 D. or over we rarely get better than 20/70 or even 20/200. Yet if marked fundus changes are absent the sight may be much better than this; and I have seen a myope of 20 D. with a vision of 15/20.

In cases with congenital inferior conus the vision is likely to be subnormal, although if the refraction be corrected carefully the vision may, in many cases, be brought up to nearly or quite the normal amount.

In myopia with marked sclero-choroiditis posterior the vision is scarcely ever normal, and if there are hemorrhages, degenerative or inflammatory changes, or a pigment spot in the macula, the sight will be damaged greatly and irreparably.

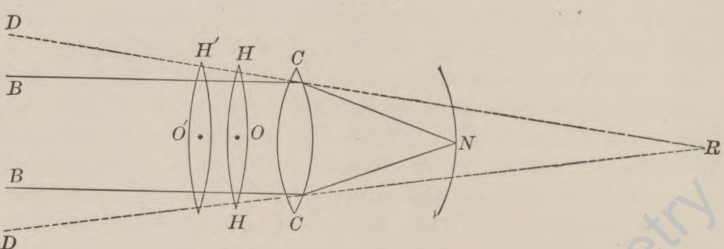
External Evidences of Myopia. If there is much myopia, the eye is often unusually prominent ("pop-eye"), and the pupil is likely to be dilated. This dilatation is attributed to the disuse of accommodation, or more probably of the convergence, for when the accommodation and convergence are relaxed the pupil regularly dilates.

Myopes also squint the eyelids in order to diminish the size of the pupil, and thus to lessen the size of the diffusion images. For the same reason myopes incline the head forward, as by so doing they partially cover the pupil, and so look through a narrower chink.

The most striking feature, and one that the laity take as a sign of nearsightedness, is the manner in which the myope holds objects close up to his eyes in order to see them, thus bringing the object within his far point. This, however, is not an absolute evidence of nearsightedness, being sometimes due merely to habit, sometimes, also, as we shall see later, being found in hyperopia of a high degree.

Hyperopia. Far Point in Hyperopia. A patient with uncorrected hyperopia sees in diffusion images. Consequently his sight is blurred, and the more so the higher the hyperopia. His natural distant vision then is poor; but, contrary to what takes place in myopia, his vision

FIG. 50.



Far point in hyperopia and course of emergent rays; correction of hyperopia by accommodation or by a convex glass. *R*, the far point of the hyperopic eye. Rays such as *D C R*, *D C R*, converging toward *R*, will by the lens *C C* (representing the cornea and crystalline lens collectively) be given the additional convergence *C N*, *C N*, so as to be focused upon the retina *N*. So, also, if *N* is illuminated and sends out rays *N C*, *N C*, back through the eye, these rays, after passing through *C C* and emerging from the eye, will take the direction *C D*, *C D*, as if they diverged from *R* (principle of conjugate foci), and will form an erect virtual image of *N*, which will appear to be at *R*. *H*, supplementary lens correcting the hyperopia. *H* may be either an actual glass lens (artificial correction) or may represent the extra bulging of the crystalline lens, produced by the accommodation (natural correction). In the former case, *H* must be of such a strength that it will make the parallel rays *B H*, *B H*, take the direction *D C R*, *D C R*, and converge to *R*, for if they are given this convergence the lens *C C* can then focus them. *R* must then be the principal focus of the lens *H*, and *O R* its focal length. If the lens *H* is moved out to *H′*, its focus must still be at *R* for it to give parallel rays the proper convergence, so that its focal length will now be *O′ R*—*i. e.*, *H′* will be a lens of longer focus—*i. e.*, a weaker lens than *H*—and yet do the same work.

for near objects is poorer still, for if his eye is naturally so weakly refracting that it cannot focus even parallel rays upon the retina, still less will it focus rays which, coming from a near object, are already divergent, and hence are just so much the more difficult to converge.

The far point of the hyperopic eye, therefore, that is, the point for which it is naturally adjusted without any effort of accommodation, is neither at far distance, as in emmetropia, nor at any nearer point, as in myopia.

Where, then, is the far point in hyperopia? Or to put the question in another way, If the eye is neither adjusted for parallel rays, as in

emmetropia, nor for divergent rays, as in myopia, for what kind of rays is it adjusted?

The answer to this question is simple. The hyperopic eye (Fig. 50) will not be able to focus the parallel rays *B B* upon the retina *N*, because it cannot give them quite the amount of convergence required —cannot, we may say, bend them quite strongly enough. But it will focus rays like *D C*, *D C*, that are already convergent to a certain amount, and which will, therefore, require a less amount of additional convergence or bending to bring them together at *N*. Rays like *D C*, *D C*, if not intercepted by the eye, would meet at some point, as *R*, back of the latter. We say, then, that the hyperopic eye is adjusted for or will, without accommodative effort, focus rays that are converging toward the point *R*, lying back of the eye. *The point R is then the far point* of the hyperopic eye.

The less the refractive power of the eye, that is, the less additional convergence it is able to impart to the rays impinging upon it, the greater must be the initial convergence of the rays *C C* which can be focused by it upon the retina. But the more *C C* converge the closer will *R* be to the eye. Hence we see, the higher the hyperopia the closer the far point.

Correction of Hyperopia. The hyperopic eye is like a camera which is out of focus because the sensitive plate is too close to the lens. Such a camera may be put in focus either by carrying the plate back to its proper place, or by leaving the plate where it is and adding to the strength of the camera lens.

In the eye both methods of compensation are possible. The *eye may lengthen*, and thus shift the retina back to a point where parallel rays will be focused upon it. Such an elongation of the eye actually takes place during the growing period, and thus the hyperopia originally present in most eyes is gradually lessened or even entirely neutralized.

This process, however, is one that goes on very gradually, and, even if it took place very much faster than it does, it could not be utilized for the rapid correction of hyperopia. This must be effected, then, by adding in some way to the deficient refractive power of the eye.

In the natural eye this is accomplished by *accommodation*. The ciliary muscle, acting in quite the same way as it does when the emmetropic eye focuses down from a distant to a near object, causes the crystalline lens to bulge, and thereby increases its refractive power by the requisite amount. The conditions, in other words, are precisely those shown in Fig. 50, where the lens *C*, representing the cornea and crystalline lens of the hyperopic eye, is supplemented by the lens *H*, which represents the additional refractive power put forth by the accommodation. The conditions that will be observed are analogous to those shown in Fig. 46.

The ability of the eye to put forth this accommodative effort, and thereby neutralize its hyperopia, will obviously diminish as the elas-

ticity of the crystalline lens diminishes. It will, therefore, decrease steadily with age. It will also be reduced by anything that impairs the power of the ciliary muscle, and will be entirely abrogated by a cycloplegic like atropine.

We may then neutralize hyperopia by a certain accommodative effort, producing an increase of refraction represented by the lens *H* (Fig. 50).

We may also produce such an increase in refraction by a *glass lens* placed directly before the eye. Theoretically, at least, it makes no difference whether *H* is actually a lens of glass or simply represents the extra bulging of the crystalline lens produced by the ciliary muscle. The hypermetrope focused for distance is, in fact, in the same position as the emmetrope focused for near. In either case, it makes no difference, so far as vision is concerned, whether the focusing is done by accommodative power, or by a glass lens, or partly by one means and partly by the other.

Naturally, then, hyperopia is corrected by accommodation and artificially by a convex lens which either neutralizes all the hyperopia or so much of it as the accommodation fails to correct.

The amount of hyperopia is usually measured in terms of the glass that completely corrects it. What, then, will be the strength of this glass?

If we revert to Fig. 50, we see that the refracting media of the eye represented by the single lens *C* will, unaided, bring to a focus upon the retina rays having the direction *D R*, *D R*—*i. e.*, rays which are already converging toward the eye's far point, *R*. The lens *H*, therefore, which will adjust the eyes for parallel rays, must be just sufficiently strong to make the parallel rays take this same direction, *D R*, and converge toward *R*. If it does this much of the work, *C* will do the rest and will focus the rays upon the retina. But a lens which makes parallel rays converge to *R* is a lens whose principal focus is at *R*; hence *the glass which completely corrects the hyperopia is a convex lens having its principal focus at the far point of the hyperopic eye.* (Compare the statement made in regard to the correction of myopia, page 82.)

Just as in the case of myopia, the strength of the correcting lens in hyperopia will *vary according to its distance from the eye.* *R* being the principal focus of the correcting glass *H*, *H R* is its focal distance. If *H* is moved away from the eye to *H'*, it will still give parallel rays the proper convergence; that is, will still correct the hyperopia, provided its principal focus is at *R*. Its focal distance now, however, is *H' R*, which is greater than *H R*. Since the strength of lenses diminishes as their focal distances increase, the lens placed at *H'* will not need to be as strong to do the same work as if placed at *H*. Or, to put the case in another way, the *effect of a convex glass in correcting hyperopia is heightened if the glass is carried away from the eye*, and *is diminished if it is carried toward the eye.* In case of a high hyperopia the difference produced in the correcting glass in this way may be considerable. Thus a patient who has had a cataract

extracted and is wearing a +11 D. will alter its strength by a whole dioptre if he shifts its position one-third inch forward or back.

It should be carefully noted that in general this increase in strength produced in a convex glass by shifting it away from the eye applies only when the glass is used for *distant* vision. When a convex glass is *used for reading, its strength is diminished* by carrying it off from the eye, provided the patient is emmetropic or but little hyperopic. If, however, his hyperopia is more than 4 D., and in some cases (*e. g.*, when he holds a book far off), if his hyperopia is no more than 3 D., he would increase the effect of his glass by carrying it off from the eye.

The true reason why many presbyopes, even though emmetropic, push their reading-glass away from the eyes when it is too weak, is that they thereby increase the size of their retinal images, although at the same time they make them less distinct.

The far point of the hyperopic eye can be determined directly from the strength of the correcting lens. Thus in hyperopia of 4 D. (or one corrected by a 10-inch lens) the far point is 10″ behind the eye.

Accommodation in Hyperopia. Near Point. The uncorrected hyperopic eye, as we have seen, has used up a certain amount of its accommodation in order to neutralize its hyperopia and to see distinctly at a distance. It has, consequently, only a residue of accommodation left for focusing down upon a near point. As compared with an emmetropic eye, therefore, having the same accommodative power, it *cannot see objects as close*. Thus a hyperope of 2 D. who has 6 D. of accommodation will, after neutralizing his hyperopia, have only 4 D. left to use in focusing upon near objects. His near point, therefore, will be at about 10″, and he can see an object that far off only by using the whole of his accommodation; while the emmetrope with the same accommodative power will still have 2 D. left when viewing an object at this distance, and by using all of his accommodation can focus down to 7″. As a hyperope and an emmetrope of the same age have about the same amount of accommodation, it follows that the hyperope will become presbyopic, that is, require glasses for reading, considerably sooner than the emmetrope will. For, as is evident from Fig. 56, his near point has receded further than the emmetrope's—in fact, is as far off as is that of an emmetrope who is a number of years older.

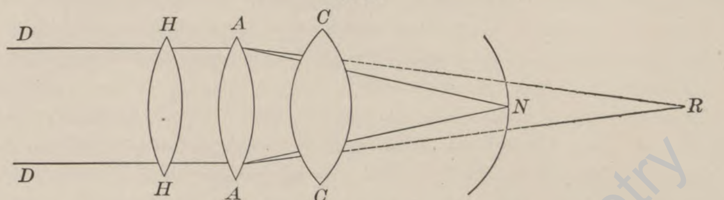
As an offset to this, it should be noted that the hyperope's accommodation, while not greater in amount than that of the emmetrope or myope of the same age, is in constant exercise and is more readily maintained. In consonance with this, we find that in hyperopes the ciliary muscle is particularly well developed.

The excess of accommodation which the hyperope has to put forth leads frequently to *accommodative convergence-excess*, which may remain as an esophoria, or may lead to a convergent squint that is at first marked only for near points, but afterward becomes pronounced both for distance and near.

In some cases of hyperopia the accommodation may be called into play so excessively as to over-correct the error, even for distance, and thus render the patient apparently *myopic*. This is especially the case if astigmatism is present or if the patient has used improper glasses. If, as sometimes happens, a concave glass is prescribed for this apparent myopia, the patient may, by continuing his constant accommodative effort, become truly myopic.

The hyperope may correct his hyperopia entirely with his accommodation, or may relax his accommodation altogether and allow us to correct the whole of his hyperopia with a convex glass. More usually, however, he allows us to correct a portion of his hyperopia with a glass, H (Fig. 51), and corrects the rest with his accommodation (A). He is, in fact, so accustomed to using his accommodation continuously that generally he cannot, no matter how we urge him, give up the effort at once when we place a convex glass before his eye, but he retains at least some of his accommodation—*i. e.*, A—at work. If, in such a case, we give a glass stronger than H by 0.50 D., the

FIG. 51.



Manifest and latent hyperopia. The hyperopic eye, instead of being corrected wholly by a convex lens, as in Fig. 50, is corrected partly by a glass lens, H, partly by the accommodation. The amount that is corrected all the time by the accommodation (represented in the figure by the lens A) is the latent hyperopia. The portion corrected by the glass lens H, but which also may, if necessary, be corrected by the accommodation, is called the manifest hyperopia. Hyperopia which cannot be corrected at all by the accommodation, but necessarily requires a glass lens to obviate it, is called absolute.

patient will at once begin to see less distinctly, for he will then, in effect, have before his eye besides the lenses A and H, which together correct his hyperopia and make him emmetropic, a lens of + 0.50 D., which will make him practically a myope of 0.50 D.

A patient as shown in Fig. 51 would be apparently hyperopic to the amount of H, although his actual total hyperopia would be equal to $A + H$. A in this case is his latent and H his manifest hyperopia. The *manifest hyperopia*, in other words, is the amount that the patient will reveal by the utmost voluntary relaxation of his accommodation, and is represented by the highest convex glass with which he can still see distinctly. The *latent hyperopia* is the remaining hyperopia, which he will not reveal in this way, because he keeps it corrected by his accommodation. The *total hyperopia* is the sum of the latent and manifest hyperopia.

The patient's vision will be practically the same whether he corrects the hyperopia entirely by accommodation, or entirely by a

convex glass, or partly by the glass and partly by accommodation. (Fig 51.) In cases, therefore, where the patient's vision is already good, because although he has hyperopia his accommodation corrects all of it, we cannot demand of a convex glass that it shall improve his sight, but only that it shall still keep the vision as good as it was before. We say, then, that he *accepts* that glass. If he accepts a convex glass, it is proof that he has hyperopia of at least that amount. If, for instance, he sees as well with a + 1.50 D. as without it, he cannot have hyperopia of simply 1 D., for in that case the addition of 1.50 D., by over-correcting the hyperopia, would render him practically myopic, 0.50 D., and would blur his vision.

We cannot say that a patient accepts a concave glass, for, as we have seen, any person with good accommodation will overcome a low concave glass and see at least as well with as without it. To prove that the patient really requires a concave glass, we should show that he sees *really better with it* than without it. If he sees simply as well with it, or apparently sees somewhat more sharply, but cannot actually distinguish any more, he is not myopic to that amount.

Varieties of Hyperopia. In hyperopia the point at which the eye focuses parallel rays lies behind the retina. This may be either because the retina is too far forward, or because the retina, being in its proper place, the cornea or lens has too little refractive power. Hence the hyperopia may be due either to changes in curvature (flattening) of the cornea, or lens (curvature hyperopia), or to changes in the density of these media (index hyperopia), or to an undue shortness of the eyeball (axial hyperopia).

Curvature hyperopia is found as a result of opacities or cicatrices of the cornea associated with flattening. This form is generally combined with astigmatism. The peculiar sort of curvature hyperopia in which one of the refractive surfaces is not simply flattened, but altogether abolished, is that produced by absence of the lens (aphakia). This in eyes previously emmetropic produces a hyperopia of 10 to 11 D.

An example of *index hyperopia* is that which develops in old age as a result of sclerosis of the lens, rendering the latter more homogeneous, and hence less refractive.

Axial hyperopia is by far the most common kind. It is also apparently the original condition subsisting in the vast majority of eyes, for examination of newborn infants has shown a very great preponderance of hyperopia, and particularly hyperopia due to a comparative shortening of the eye.

During the growing period of childhood and youth the eye becomes steadily longer, so that the *hyperopia grows less and less.* It may thus be transformed into emmetropia, or possibly go over into myopia. This process of elongation regularly ceases at the age of twenty-two years, so that if any hyperopia is left then, it remains stationary thereafter.

One millimetre of shortening of the eyeball corresponds to about

3 D. of axial hyperopia, so that, for instance, an eye which is 2 mm. shorter than normal will be hyperopic 6 D.

In axial hyperopia the shortening affects chiefly that portion of the eye back of the lens, although especially in high hyperopia the cornea may be flat and the anterior chamber shallow.

A pathological form of axial hyperopia is produced by exudates pressing the retina forward, by detachment of the retina, and by the presence of tumors behind the eyeball, which indent the latter.

Amount of Hyperopia. Two classes of cases may be recognized. In one the hyperopia does not exceed 5 D., and both exterior and fundus present no abnormalities. Such hyperopia may be called normal. Hyperopia of over 5 D. may also be normal, but very frequently in hyperopia of this amount we find evidences of arrested development, such as albinism, microphthalmus, a small cornea, an imperfectly developed fundus, etc. Such hyperopia is abnormal.

Vision in Hyperopia. The sight in hyperopia depends upon the patient's ability to correct his error by means of his accommodation. A young person who has an abundance of accommodative power and no inordinate amount of hyperopia will see distinctly and without any great difficulty both for distance and near. As he grows older and his accommodation diminishes, he will be able to see distinctly only by the expenditure of more and more effort. Finally, a point is reached where near vision is effected with difficulty and strain, although distant vision is still easy. Later, distant vision becomes difficult and near vision impossible, and, last of all, his accommodation fails him even for distance, and he sees poorly at all ranges.

The hyperopia that the patient can fully correct by his accommodation is called *facultative;* one which he can correct only by calling into exercise an amount of accommodative power which induces a convergent squint is called *relative;* and one which he cannot correct at all by his accommodation is called *absolute.*

The period at which this failure of accommodation to compensate for hyperopia occurs—*i. e.*, at which the latter becomes absolute—varies a good deal, according to the general build and constitution of the patient, the kind of work to which his eyes are subjected, etc. In general, persons with less than 2 D. hyperopia, whose eyes are not excessively taxed in near work, will not suffer serious inconvenience until past twenty-five or thirty years, when they will generally require a glass for reading, but will not absolutely require one for distance for perhaps ten or twelve years later.

A hyperope of 2 to 3 D. will probably experience some annoyance in youth if he uses his eyes much for studying, and after the age of twenty will usually require a glass for near and probably, also, for distance.

Hyperopes of 3 to 4 D. usually require glasses in childhood, both for distance and near, as, while they still can see distinctly, they do so at the expense of considerable effort, and hence develop either an asthenopia or a convergent squint.

Hyperopes of more than 5 D. rarely can overcome their defect by accommodative effort, and do not often make an attempt to do so. Such patients simply have poor vision and do not have asthenopia. Their sight, even with the best correction, is often subnormal (20/40 to 20/200 or less).

External Evidences of Hyperopia. Hyperopes often have small *pupils*. This is believed to be occasioned by the excessive accommodative effort, or rather the excessive convergent effort, that such patients make, as the acts of accommodation and convergence are associated always with contraction of the pupils.

The front of the eyeball in well-marked hyperopia often appears flattened and the anterior chamber may be shallow.

Most hyperopes tend to hold their books rather far away on account of the recession of their near point. Now and then, however, a patient will be found with hyperopia, particularly when the hyperopia is of high degree, who holds his book very close to him, and on that account is thought to be myopic. This mistake is the more natural as such a patient often has poor sight for distance, because his hyperopia is too great to be neutralized by the accommodation. Of course, his vision for near is still poorer, and the more so the closer he brings objects to his eyes. Hence he is not really like a near-sighted person, who by bringing objects closer makes them more distinct. The hyperope brings the object nearer to make it appear larger, and hence more readily distinguishable, even if it is more blurred.

Astigmatism. Varieties of Astigmatism. Disposition of the Meridians in Astigmatism. In astigmatism the different meridians of the eye have different refractive powers, so that each focuses the rays of light differently from the meridian adjoining. If the change in refractive power takes place uniformly and by regular degrees from one meridian to another, so that each meridian in succession refracts a little more strongly than the one before it; and if, furthermore, the refraction in one meridian shows no great or sudden changes from its centre to its periphery, the astigmatism is called *regular;* and in the contrary case, *irregular*.

That kind of astigmatism produced by difference in refraction between the central and peripheral part of any one meridian of the eye is called *meridianal aberration*.

In most cases of regular astigmatism the meridian that refracts the most highly, that is, focuses the rays of light most quickly, is vertical or within 30° of the vertical, and such astigmatism is said to be *with the rule* or *direct*. The next most frequent variety, called astigmatism *against the rule*, or *inverse* astigmatism, is that in which the most highly refracting meridian is horizontal, or within 30° of the horizontal. Less often met with is *oblique* astigmatism, in which the meridian of greatest refraction lies at from 30° to 60° from the vertical.

In regular astigmatism the meridians of greatest curvature (prime meridians) are usually *symmetrically disposed* in the two eyes; that

7

is, both are either just vertical or just horizontal, or both are inclined by an equal amount to the temporal or both to the nasal side of the vertical. Much more rarely the meridians are *parallel* in the two eyes. It is also uncommon to find them *neither symmetrical* nor *parallel.* Very rarely the prime meridians in the two eyes are at *right angles* to each other.

In regular astigmatism the meridian which refracts the most strongly almost always lies at right angles to the meridian which refracts the least. These two are called the *principal meridians.* The amount of astigmatism then is measured by the difference in refraction existing between these two.

Etiology and Development of Astigmatism. With regard to the etiology, we should distinguish between *idiopathic* or *primary* astigmatism, which is not, and *secondary* or *pathological* astigmatism, which is due to disease of the eye.

A certain amount (0.25 to 0.50 D.) of primary astigmatism may be regarded as *physiological* in that at least that amount is found in nearly every eye. Physiological astigmatism is regularly present both in the cornea and in the lens, and in both situations is partly regular and partly irregular.

The higher degrees of primary astigmatism (over 1 D.) are mainly of the regular variety. Astigmatism of 1 to 2.50 D. is very frequent, although not so much so as to be regarded as normal. Astigmatism of 2 to 4 D. is fairly common, while primary astigmatism of more than 5 D. is rare.

This non-physiological primary astigmatism, like the physiological, is usually present both in the cornea and lens, although the cornea is apt to play a much larger part in its production. Frequently corneal astigmatism with the rule is combined with lenticular astigmatism against the rule, so that the resulting total astigmatism is less than the corneal astigmatism. In other cases, but less often, the lenticular astigmatism adds to instead of correcting that of the cornea; the astigmatism of both lens and cornea being then usually inverse. Again, the combined corneal and lenticular astigmatism is often such that the meridian of greatest refraction of the eye as a whole does not coincide with the meridian of the greatest curvature of the cornea. These variations are important in estimating the value to be ascribed to the findings obtained by the ophthalmometer.

In the cornea primary astigmatism is due to unequal curvature. In the lens primary astigmatism may also be due to unequal curvature, but more usually to the fact that the lens is tilted somewhat, and, furthermore, that it is built up of separate fibrillæ of different densities. These fibrillæ are grouped so as to form star-like figures on the front and back surfaces of the lens. Rays of light passing from one fibrilla to another are refracted somewhat irregularly, and thus a certain amount of irregular as well as of regular astigmatism is produced.

Primary astigmatism is either congenital, or probably much more often develops in early childhood. Once developed, it remains on the whole practically stationary during childhood, youth, and early manhood. Changes do take place in it from time to time, but they are inconstant and usually slight, and there is no special rule governing the way in which they develop. After middle life, however, there is a tendency for astigmatism to change its character. Astigmatism with the rule tends to become less, or is sometimes converted into astigmatism against the rule, and astigmatism against the rule often develops where no astigmatism existed before, so that in old age astigmatism against the rule is the most common variety.

Secondary or *pathological astigmatism* occurs both in the cornea and in the lens. In the cornea it is due to the unequal flattening or bulging produced by cicatricial contraction, such as takes place in the healing of wounds and ulcers, or by faceting of the cornea, or by non-inflammatory protrusion, as in keratoconus. Astigmatism of this kind, while mainly irregular, is associated often with a large amount of regular, and therefore, corrigible astigmatism. This is particularly true of the astigmatism produced by cicatricial contraction after operation wounds in the cornea. A marked example of this is the regular inverse astigmatism of 2 to 8 D., quite uniformly found after cataract extraction.

Secondary lenticular astigmatism, regular and irregular, is produced by variations in its curvature (that due to bulging of a dislocated lens, for instance), or by alterations in the density (nuclear sclerosis or other changes due to advanced age).

Pathological astigmatism will remain stationary or change according to the behavior of the process producing it. Thus a corneal astigmatism produced by a cataract extraction regularly decreases during the three or four months following the operation (often diminishing several dioptres) until the process of cicatrization is completed, when it remains stationary. Astigmatism due to keratoconus often goes on increasing for years, either continuously or at intervals.

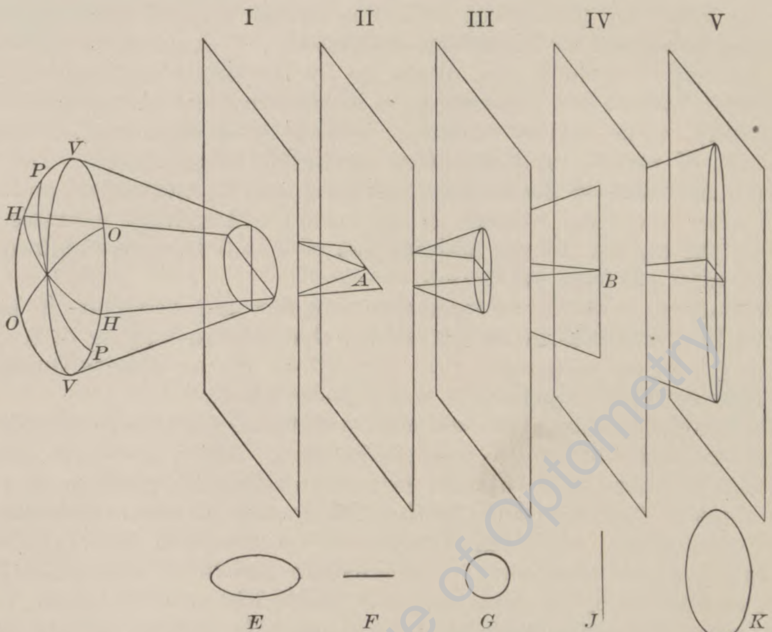
Absolute Refractive State in Astigmatism In regular astigmatism the rays passing through meridian No. 1 come to a focus in front of those passing through meridian No. 2, next adjoining, and these again in front of those passing through meridian No. 3, etc. But this fact tells us only that one meridian refracts more strongly than the other, but does not tell us what the *absolute refraction* of each meridian is. This will depend entirely upon where the retina is situated.

If (Fig. 52) we have a case of astigmatism with the rule, rays coming from a distant point of light will pass through the vertical meridian *V V* of the cornea and lens, and will be focused at some point, *A*. Rays passing through the horizontal meridian *H H* will be focused further back at some point, *B*. Rays passing through the various oblique meridians, as *O O*, will be focused at some point between *A* and *B*. The space from *A* to *B* is called the *focal interval*. In the given case we will suppose this to measure 2 mm.

If now, the retina is at I, 1 mm. in front of *A*, all the meridians of the eye will be hyperopic, and the horizontal meridian most so. In fact, the vertical meridian will be hyperopic 3 D., because, so far as this meridian is concerned, the eye is 1 mm. too short, while the horizontal meridian will be hyperopic 9 D., because so far as it is concerned the eye is 3 mm. too short. The difference in refraction between the two meridians—*i. e.*, the astigmatism—is therefore 6 D., or the eye has a hyperopia of 3 D. combined with an astigmatism of 6 D.

If now, the retina recedes to II, rays passing through the vertical meridian have come to a focus, and, so far as this meridian is concerned,

FIG. 52.



The absolute refraction in astigmatism. Form of diffusion images. *A*, focus of rays passing through vertical meridian *V V*. *B*, focus of rays passing through horizontal meridian *H H*. *A B*, focal interval. I, compound hyperopic astigmatism; II, simple hyperopic astigmatism; III, mixed astigmatism; IV, simple myopic astigmatism; V, compound myopic astigmatism. *E, F, G, J, K*, forms of diffusion images at I, II, III, IV, V, respectively.

the eye is emmetropic. In fact, if by using a vertical slit we shut off all rays but these, the eye would be adjusted accurately for distance; but for rays passing through all other meridians the eye is hyperopic, and, in fact, for the horizontal meridian is hyperopic 6 D. We have, therefore, still a difference between the meridians, or an astigmatism, of 6 D., although now one meridian is emmetropic. This condition is called *simple hyperopic* astigmatism, while the condition obtaining while the retina is at I is called *compound hyperopic* astigmatism. It is evident that the compound differs from the

simple astigmatism merely in adding the same amount of hyperopia (in this case 3 D.) to all meridians alike.

Suppose, now, the retina recedes to III two-thirds of a millimetre back of *A*. In the vertical meridian the eye will now be myopic 2 D., because the rays passing through this meridian will come to a focus two-thirds of a millimetre in front of the retina. In the horizontal meridian, however, the eye will be hyperopic 4 D., since the retina lies one and one-third millimetres in front of *B*. The difference in refraction between the horizontal and vertical meridians, namely, the astigmatism, is still 6 D. It is evident that the only change produced in passing from II to III is to make all the meridians of the eye alike more refractive by 2 D. The vertical meridian which was emmetropic and those meridians adjoining which were hyperopic less than 2 D. have now become myopic. Two oblique meridians, such as *O O* and *P P* on either side of *V V*, which were hyperopic 2 D., have become emmetropic, and all other meridians remain hyperopic. Such a condition, in which some meridians are hyperopic and some myopic, is called *mixed* astigmatism.

If the retina recedes to IV, the horizontal meridian is now emmetropic, while the other meridians are myopic, and the vertical meridian is myopic 6 D. The astigmatism is still 6 D. Such an astigmatism is called *simple myopic* astigmatism. It is evidently evolved from the simple hyperopic astigmatism indicated at II, by rendering all meridians alike 6 D. more refractive.

Finally, when the retina is at V, one millimetre behind *B*, all the meridians will be myopic, the vertical one 9 D., and the horizontal 3 D. The astigmatism will still be 6 D. This condition is known as *compound myopic* astigmatism.

We see, then, that a man starting with a certain amount of hyperopic astigmatism may by gradually elongating his eye, or by equally increasing its refractive power, *acquire all varieties of astigmatism in succession, but will leave the amount of astigmatism unchanged.* And, as a matter of fact, this conversion of one variety of astigmatism into another takes place very frequently either through gradual lengthening of the eye during the growing period, or through uniform increase of the refractive power produced by accommodation. It may also be brought about by an artificial increase or decrease of the refraction produced by glasses.

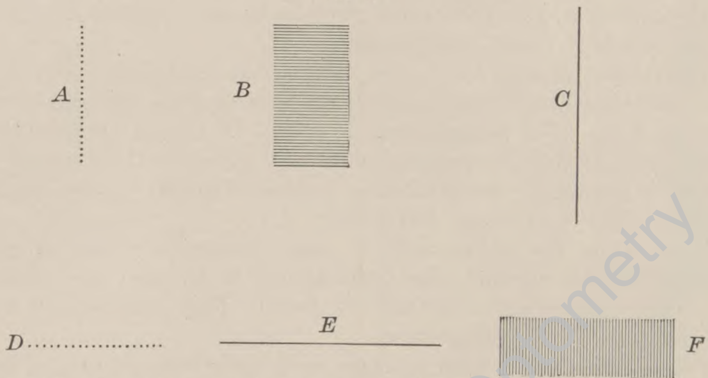
Vision in Astigmatism. Diffusion Images. Irregular astigmatism makes objects look *distorted*, just as imperfectly made window glass does. If the astigmatism is high, the distortion is extreme, and even very low degrees of astigmatism, especially when present in the crystalline lens, produce considerable deformity of minute objects. In fact, the reason why stars look like stars instead of like infinitesimal points of light, as they should do, is because of this irregular astigmatism of the lens due to the star-like deposition of its fibrillæ.

In regular astigmatism the amount of distortion and blurring produced depends upon the *diffusion images*. Inspection of Fig. 52

will show that at I the diffusion image of a point will be a horizontal ellipse, *E;* at II a horizontal line, *F;* at III a horizontal oval, *G* (which as the retina recedes will be converted first into a circle, then into a vertical oval); at IV a vertical line, *J;* and at *V* a vertical oval, *K.*

At II the image of a point is a fine horizontal line, and the image of a horizontal line, which is nothing but a series of points strung along horizontally, will be a series of faint horizontal lines. These by successive overlapping will reinforce each other and form a broad, distinct horizontal line a little thickened and hazy at its ends. (Fig. 53.) On the other hand, a vertical line, being composed of a row of points, one above the other, will form an image made up of a set of faint horizontal lines one above the other, and will thus form a dim, hazy, more or less broadened band. Hence it is seen that, although the

FIG. 53.



Images of a line formed in astigmatism. *A*, a vertical line consisting of a vertical row of points; *B*, its image when, as in Fig. 52, II, the vertical meridian of the eye is emmetropic; *C*, its image when, as in Fig. 52, IV, the horizontal meridian of the eye is emmetropic. *D*, a horizontal line consisting of a row of points side by side; *E*, its image when the vertical meridian of the eye is emmetropic; *F*, its image when the horizontal meridian of the eye is emmetropic.

vertical meridian is emmetropic, the image of the vertical line is very indistinct and the image of a horizontal line is quite sharp. All objects will appear drawn out into horizontal lines.

At IV all objects will appear drawn out into vertical lines, and consequently, although it is now the horizontal meridian that is emmetropic, the horizontal lines are seen most indistinctly and vertical lines most clearly.

In other words, in simple astigmatism, myopic or hyperopic, *those lines are seen most distinctly which run at right angles to the emmetropic meridian.*

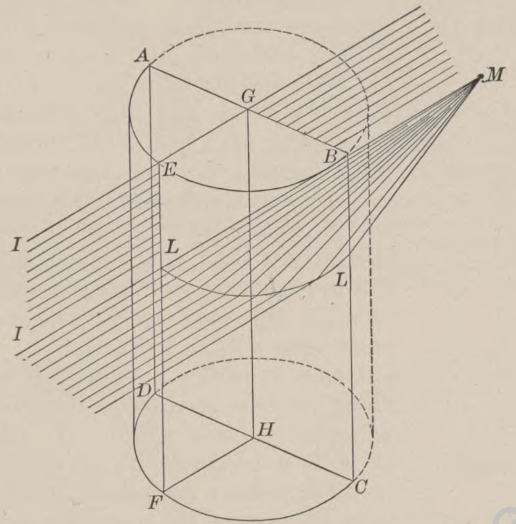
It will also be readily seen that when the retina is at II a square would look like an oblong drawn out laterally, with the tops distinct and the sides blurred; a circle like a horizontal oval, etc. If the retina were at IV, the reverse effect would be produced. In fact, as

the astigmatism changes from hyperopic to mixed, and then to myopic, the character of the distortion will change, so that sometimes one and sometimes another set of lines will appear distinct, and objects will seem to be drawn out now in one, now in another direction.

Besides these distortions of shape, astigmatism produces *monocular diplopia*, objects seeming to have close to them a faint shadow of themselves. The position of this double image will vary according to the direction of the principal meridians.

Accommodation and Astigmatism. As we have noted, accommodation frequently alters the character of the astigmatism, changing it from hyperopic to mixed, and then to myopic, to suit the needs of the patient's vision, but it rarely changes its amount, at least

FIG. 54.



A convex cylinder. *E F*, axis of cylinder. Rays *I I* passing through the cylinder in the plane of this axis go on without undergoing refraction. Rays *K K* striking the cylinder in a plane perpendicular to the axis are converged to *M*.

materially. We do, however, find in a certain number of cases that the astigmatism is greater under atropine than without it, leading us to suppose that the accommodation had concealed a certain amount of it. And occasionally the astigmatism becomes less, and even disappears when atropine is instilled, from which we should infer the astigmatism to be a spurious one, produced by unequal accommodative effort.

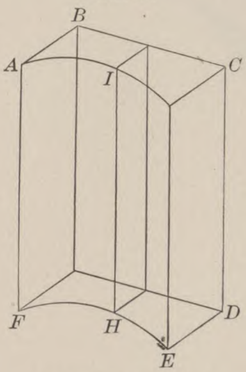
Correction of Astigmatism. Astigmatism may then in part, but only to a very slight extent, be corrected by accommodation. More often accommodation, while leaving the amount of astigmatism unchanged, affects the vision favorably by adjusting the eyes for different sets of lines. An eye, for instance, which is in the condition denoted by Fig. 52, II, will see horizontal lines distinctly. But in

reading and many other visual acts it is important to see vertical lines distinctly. This the patient may do by exerting his accommodation so that his condition is changed to that of IV. He will then see vertical lines distinctly, and, if it is also important to see horizontal lines distinctly, he may do this by narrowing the fissure of the lids a little so that he may look through a horizontal chink.

We may also correct astigmatism by means of *cylinders*.

Cylindrical glasses are either convex (positive) or concave (negative). The *convex* cylindrical glass shown in Fig. 54 is a slice, *A B C D*, taken from a cylinder. The line *E F*, parallel to the axis of the original cylinder, is called the *axis* of the glass. Rays such as *I I* entering the cylinder along its axis *E F*, pass through it in the plane *E F G H*, and will undergo no refraction, because the lines they encounter are not curved but straight. Rays, on the other hand, as *K K*, entering the cylinder in the plane perpendicular to its axis, will be refracted just as in a spherical lens, for they will strike a line, *L L*, whose curvature is a circle, and they will therefore come to a focus at a point, *M*, behind the glass. Rays passing through the glass obliquely to the axis will also be refracted, but not so much as *K K*, because they strike a line which is less curved than *L L*, and they will hence be focused at a point behind *M*. The more inclined the rays are to the axis *E F*, the more they will be refracted.

Fig. 55.



A concave cylinder.

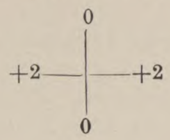
Hence a convex cylinder is a glass which along its axis produces no refraction, that is, acts like a plane glass. At right angles to its axis it produces its maximum effect, and in intermediate meridians produces an effect which increases with the inclination of the meridian to the axis.

A concave cylindrical glass, *A B C D E F* (Fig. 55), represents the cast of a convex cylinder. Its axis would be *I H*. It behaves precisely like a convex cylinder, except that it acts like a negative (dispersing) instead of a positive (converging) lens.

In naming cylinders, it is necessary to give their sign, strength, and direction of their axes. Their *strength* is that of their meridian of greatest refraction (+ or —), that is, the meridian at right angles to their axis. The *direction* of the axis is denoted in a number of ways, of which three are indicated in Figs. 56, 57 58. Whatever method is used, it is always best to have on prescription blanks a diagram like one of the above, and to indicate by a stroke on the diagram the precise direction of the axis. This acts as a useful check and guards against mistake as to the intent of the prescriber.

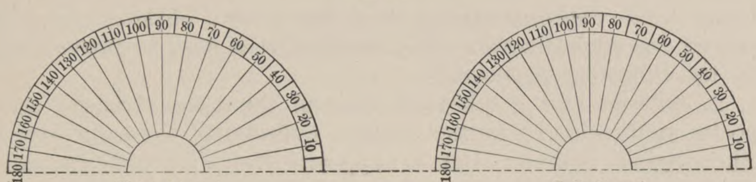
A glass which is compounded of a spherical glass and a cylinder is called a *sphero-cylinder*.

In testing refraction, it is important to be able to add and subtract cylinders with facility. How this is done is shown best diagrammatically. A +2 cylinder axis 90°, for instance, is denoted by



in which the glass is supposed to be seen face on. The vertical meridian (axis) is 0 D., the horizontal meridian has the effect of

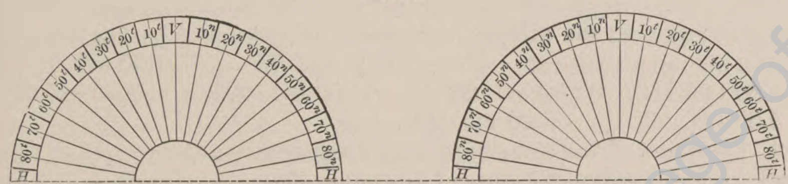
Fig. 56.



R (*right eye*) *L* (*left eye*)

Ordinary or parallel method of indicating the axis of cylinders or the direction of prisms. In each eye the position of the axis of the cylinder is denoted by the angle it makes with the horizontal, this angle being always measured from the right-hand side of the observer (left-hand side of the patient). The numbering thus runs through 0° to 180°, starting at the nasal side in the right eye and at the temporal side in the left. The horizontal is always denoted by 180° ; vertical is 90°.

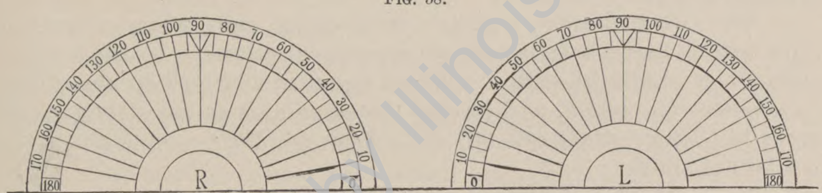
Fig. 57.



R (*right eye*) *L* (*left eye*)

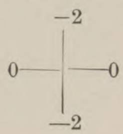
Bisymmetrical method of indicating the axis of cylinders. In each eye the position of the axis of the cylinder is denoted by the angle it makes with the vertical meridian V (= o°), either on the nasal or on the temporal side, and is written as follows: 5n = 5° n, 5t = 5° t, etc., down to *H* = 90° (horizontal).

Fig. 58.

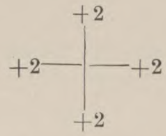


Symmetrical method of indicating the axis of cylinders. In each eye the position of the axis of the cylinder is denoted by the angle it makes with the horizontal, the angle always being measured from the nasal to the temporal side. The numbering thus runs from 0° to 180°, from right to left in one eye, and from left to right in the other. Horizontal is denoted by 180°, vertical by 90°. The numbering for the right eye is the same in the symmetrical and the parallel methods (Fig. 56), while for the left eye the numbers run just opposite.

a +2 D. sph., and the intervening meridians have effects ranging from 0 D. to +2. A —2 D. cylinder 180° would be denoted by



and a +2 sph. by



Using these diagrams and noting the results obtained by superimposing one such diagram upon another, it is easy to prove graphically the following propositions:

1. A cylinder and a spherical glass of the same strength and of opposite signs added together make a cylinder of the same strength as the original cylinder, but of opposite sign and with its axis at right angles to it. Thus +1.00 sph. $\bigcirc$ —1.00 cyl. 90° = +1.00 cyl. 180°; —2.00 sph. $\bigcirc$ +2.00 cyl. 70° = —2.00 cyl. 160°.

2. Two cylinders of the same sign and same axis added together make a cylinder equal to the sum of the two. Thus +2.00 cyl. 90° $\bigcirc$ +3.00 cyl. 90° = +5.00 cyl. 90°; —1.00 cyl. 20° $\bigcirc$ —2.00 cyl. 20° = —3.00 cyl. 20°.

3. + and — cylinders of the same axis added together make a cylinder equal to the difference between the two and having the sign of the stronger cylinder. Thus +2.00 cyl. 75° $\bigcirc$ —1.00 cyl. 75° = +1.00 cyl. 75°; +2.00 cyl. 60° $\bigcirc$ —4.00 cyl. 60° = —2.00 cyl. 60°; +3.00 cyl. 90° $\bigcirc$ —3.00 cyl. 90° = 0.

4. Cylinders of the same sign and of equal strength crossed at right angles make a spherical glass of the same strength. Thus +2.00 cyl. 90° $\bigcirc$ +2.00 cyl. 180° = +2.00 sph.; —1.00 cyl. 50° $\bigcirc$ —1.00 cyl. 140° = —1.00 sph.

5. Conversely, any spherical glass may be represented as equivalent to two cylinders of the same strength and same sign crossed at right angles.

6. Any two cylinders, *a* and *b*, of the same sign, when crossed at right angles make a spherical glass equal in strength to the weaker cylinder *a* combined with a cylinder equal in strength to the difference between *a* and *b*, and having the same axis as the stronger cylinder *b*. Thus +2.00 cyl. 90° $\bigcirc$ +3.00 cyl. 180° = +2.00 sph. $\bigcirc$ +1.00 cyl. 180°.

7. A + cylinder of strength *a* crossed at right angles upon a — cylinder of strength *b* makes a glass which may be represented also by a + spherical of strength *a* combined with a — cylinder of strength $(a + b)$ and having the axis of *b*; or else by a — spherical of strength *b* combined with a + cylinder of strength $(a + b)$ and having the

axis of *a*. Thus +2.00 cyl. 90° $\supset$ —3.00 cyl. 180° = +2.00 sph. $\supset$ —5.00 cyl. 180° or = —3.00 sph. $\supset$ +5.00 cyl. 90°.

The above propositions show that when to a + cylinder we add a + cylinder in the same axis or a — cylinder at right angles, we *add* just so much to the cylindrical effect; and when we add to it a — cylinder in the same axis or a + cylinder at right angles, we *subtract* just so much from the cylindrical effect.

The student of refraction should work out these propositions by means of the diagrams above indicated, and by the same means should solve all kinds of problems connected with cylindrical combinations until he can do such problems at once without stopping to think or calculate. Until he can do this, he will be considerably handicapped in all phases of his refraction work, and will scarcely be able to do it with either rapidity or precision.

Occasionally prescriptions are seen in which two cylinders are ordered at *oblique angles* to each other. Such a combination can always be represented by one cylinder combined with a spherical glass. To compute the strength of the axis of the cylinder and the strength of the spherical glass in these cases requires a trigonometrical calculation. It is only when cylinders are combined with their axes either together or at right angles that the equivalent glass (sphero-cylinder, cylinder, or sphere) can be found by the above simple rules.

A spherical lens may be made to have the *effect* of *a cylinder* by tilting it either sideways or up and down.

Application of Cylinders to Correct Astigmatism. It is evident, by referring to Fig. 52, that we may change condition I into condition II by correcting the 3 D. of hyperopia with a +3 D. spherical glass. This will render the vertical meridian emmetropic, and will reduce the hyperopia of the horizontal meridian from 9 D. to 6 D. So also we may alter condition V into condition IV by means of a —3 D. spherical glass, which will diminish the refraction of all the meridians by that amount, and will make the horizontal meridian emmetropic and the vertical meridian myopic 6 D. Lastly, we may change condition III into condition II by adding a —2 D., and into condition IV by adding a +4 D. spherical glass. Thus any form of astigmatism may be converted into simple hyperopic or simple myopic astigmatism by means of a spherical glass. It remains, then, to consider how we may correct simple astigmatism.

To correct the simple hyperopic astigmatism shown in Fig. 52 at II, we should use a +6 D. cyl. ax. 90°. This will place the inactive axis of the cylinder opposite the emmetropic vertical meridian of the eye. Opposite the horizontal meridian, which is hyperopic 6 D., we place that portion of our glass which acts like a +6 sph. Opposite the other meridians of the eye which gradually decrease in refractive power from emmetropia to a hyperopia of 6 D., we place a glass which gradually increases in refractive power from 0 to +6 D. Each meridian, therefore, in turn has its deficiency in refractive power corrected, and thus is made emmetropic.

So, also, to correct the condition shown in IV, we should use a —6 cyl., ax. 180°. This would correct the myopia of the vertical and of each succeeding meridian, and leave the horizontal meridian emmetropic.

A little consideration will show that to correct any case of regular astigmatism we may proceed in either one of two ways. Either we may place before the eye a *convex* cylinder of a strength equal to the amount of astigmatism, with its axis in the meridian of greatest refraction, and then combine this with a spherical glass that will correct the refraction of that meridian; or we may use a *concave* cylinder of the same strength, with its axis in the meridian of least refraction, and add a spherical glass that will correct the refraction of this meridian. Thus condition I may be corrected either by +3 sph. $\bigcirc$ +6 cyl., ax. 90°, or +9 sph. $\bigcirc$ —6 cyl. ax. 180°—*i. e.*, we may regard condition I as being condition II, in which the retina has been brought forward 1 mm., or in which all of the meridians of the eye have alike been made 3 D. more hyperopic; or we may regard it as condition IV, in which the retina has been advanced 3 mm., so that all meridians alike have been made more hyperopic by 9 D. So condition II may be corrected either by +6 cyl., ax. 90°, or by +6 sph. $\bigcirc$ —6 cyl., ax. 180°; condition III, either by —2 sph. $\bigcirc$ +6 cyl. 90° or by +4 sph. $\bigcirc$ —6 cyl. 180°; condition IV, by —6 cyl. 180° or by —6 sph. $\bigcirc$ +6 cyl. 90°; and condition V, by —3 sph. $\bigcirc$ —6 cyl. 180° or by —9 cyl. $\bigcirc$ +6 cyl. 90°.

In general, when possible, we prefer to combine a + spherical with a + cylinder or a — sphere with a — cylinder, rather than combine spheres and cylinders of opposite signs.

Some prefer to correct a case of mixed astigmatism by *crossed cylinders*, prescribing, for instance, in case III, +4 cyl. 90° $\bigcirc$ —2 cyl. 180°. There is no special advantage in this prescription over the equivalent prescription, +4 sph. $\bigcirc$ —6 cyl. 180°, or —2 sph. $\bigcirc$ +6 cyl. 90°, and the latter usually are ordered.

Anisometropia. Anisometropia is a condition in which one eye differs in its refraction from its fellow. Slight degrees of it are not uncommon, but high degrees are rare. In some cases the difference may be ascribed to pathological influences which have affected differently the two eyes, but more often no cause can be ascertained.

The more ametropic eye often has very poor sight, and frequently squints, divergent strabismus being particularly common. Apart from true squint, hyperphoria and other anomalies are more frequent in anisometropic than in other eyes.

SYMPTOMS OF REFRACTIVE ERRORS.

Varieties of Symptoms Common to all Errors. The main direct symptoms that refractive errors produce are impairment of sight and

asthenopia. By *asthenopia* is meant a sense of fatigue and discomfort in the eyes, produced by use. There may be fatigue alone (*asthenopia simplex*), or the fatigue may be accompanied by pain in the eyes (*asthenopia dolens*), pain in the head (*asthenopia cephalalgica*), or conjunctival irritation manifested by lacrymation and congestion (*asthenopia irritans*). This last may lead to actual conjunctivitis and blepharitis.

A peculiar form of asthenopia (*panorama asthenopia*) is that in which a sense of vertigo, confusion, and uncertainty is induced by looking at moving, and especially at brilliantly illuminated objects. Persons affected with this symptom often suffer considerable distress from shopping in a crowded store, or walking in a crowded street, or from watching processions, ball matches, or plays.

Asthenopia may often be relieved by ceasing to use the eyes, provided this is done as soon as the symptoms manifest themselves ; but in many cases it persists for hours, or even until the following day. In some cases, again, asthenopic symptoms do not appear until the following day.

The causes of asthenopia from refractive errors are partly undue accommodative effort (*accommodative asthenopia*); partly the pressure on the eye and the congestion of the lids produced when the latter are squeezed together in order to narrow the palpebral fissure, and thus enable the patient to see more distinctly (*tarsal asthenopia*); partly a strain imposed upon the external muscles of the eye (*muscular asthenopia*); and partly over-sensitiveness of the retina (*retinal asthenopia*). It is enhanced by anything (bright lighting, etc.) that makes unusual demands upon the eyes.

It should be noted that asthenopia is by *no means always due to refractive errors*. It is often due to muscular anomalies, particularly convergence-insufficiency and hyperphoria, and, in not a few instances, to nasal affections, such as pressure in the region of the middle turbinates. Some of the most severe and obstinate cases I have seen have been due to this latter cause. Such cases are apt to be associated with marked eyeache and occipital pain. Again, asthenopia is often due to neurasthenia and other conditions marked by enfeeblement of the nervous system.

Besides the headache, eyeache, etc., that, as noted above, may accompany asthenopia, or may also occur independently of it in connection with refractive errors, we find occasionally *other symptoms*, such as nausea, interference with nutrition, various paræsthesiæ, etc. That graver reflex manifestations (epilepsy, chorea) ever are caused by refractive errors, is doubtful, although there is no question as to the propriety of correcting such errors in persons afflicted with these neuroses, and thus relieving them of at least one source of strain.

Symptoms in Myopia. Turning now to the individual refractive errors, we find that in myopia, when not of inordinate amount, the main symptom is the *impairment of vision* for distance, which is greater

in proportion to the degree of nearsight. The sight for near, on the other hand, is very distinct, objects appearing not only clearer cut, but also magnified, so that the patients are able to do very fine work (sewing, embroidery). At the same time, objects are held very close; and if binocular vision is maintained, the excessive convergence effort may produce asthenopia. The blurring in distant vision usually causes no discomfort in ordinary myopia, but in low myopia, where the blurring is slight and the patient makes constant fruitless efforts to see distinctly, a disagreeable asthenopia may develop, which is relieved by a concave glass.

In high myopia, particularly when there is advanced sclero-choroiditis posterior, there may be aching *pain* in the back of the eyeball and symptoms of *retinal irritation,* such as flashes of light, asthenopia from hyperæsthesia of the retina, etc.

In myopia *muscæ volitantes* are frequent. This may be no more than a normal phenomenon, which is accentuated here simply because the myope sees in a sort of haze or cloud upon which he readily projects the floating black specks. In high myopia the floating bodies are larger, and are evidences of liquefaction of the vitreous itself due to disease of the fundus.

Symptoms in Hyperopia. In hyperopia of moderate degree the sight is good for distance and near; and if the accommodation is effective, is performed without strain, and hence without asthenopia. When the accommodative power is low as compared to the amount of hyperopia, *asthenopia* develops for near work, and later for distance also. In high degrees of hyperopia the *sight begins to be blurred* for near, and, as accommodation diminishes, for distance too. At first the blur itself is momentary only, the sight clearing up as soon as the patient rests the eyes; but later on, the interference with sight becomes more and more constant. In very high hyperopia the patient never sees distinctly either for distance or near.

As soon as the vision becomes indistinct the asthenopic symptoms generally cease, because the patient no longer tries to accommodate. *Headaches, eyeaches,* etc., are not very common in hyperopia uncomplicated by astigmatism.

Symptoms in Astigmatism. In astigmatism the sight is blurred by the characteristic diffusion images. There is more or less *distortion* of objects looked at, and there may be *monocular diplopia.* Moreover if the patient be using his accommodation to adjust his vision for different lines in succession, he will see first upright, then horizontal lines distinctly. This produces in him the effect of an *apparent movement* of objects, so that wheel-like figures appear to revolve and check patterns to dance. The result is often a sensation of *vertigo* and *nausea.* For the same reason *panorama asthenopia* is common in astigmatism. Ordinary *asthenopia* combined with *headache* and *eyeache* is frequent, particularly when the eyes are used for near work, like reading or sewing, which requires accurate definition of the outlines of objects. In low astigmatism, *conjunctival irritation* and *blepharitis* are not in-

frequent. These are doubtless due to the frequently repeated strong contraction of the lids made in order to narrow the palpebral aperture and thus enhance the clearness of sight. This contraction leads to a more or less permanent venous congestion.

GENERAL REMARKS ON METHODS OF EXAMINING FOR REFRACTIVE ERRORS.

Varieties of Tests. Routine of Examination. We have various means for examining the eye for refractive errors. In some of the tests used, such as the tests of visual acuity, trial case tests, and astigmatic charts, we are dependent upon the patient's statements of what he sees. They are hence called *subjective tests*. In others, such as those made with the ophthalmometer, the ophthalmoscope, and, skiascopy, we are independent of the patient's statements. These are the *objective tests*.

Usually both subjective and objective tests should be combined in making an examination. Sometimes the subjective tests are inapplicable, as in children and illiterates; but whenever they can be applied, they should, in general, constitute our court of last resort, and the evidence derived from them should outweigh that of the objective methods. In other words, the correcting-glass determined on is that which gives the patient the best vision, and not that which is apparently shown by the ophthalmoscope and shadow test.

Practitioners differ a great deal in their estimates of the relative value of the tests and the way in which they should be applied in practice. It probably makes little difference what routine we adopt, provided it be systematically pursued and contains a sufficient number of tests to act as checks upon each other, and thus ensure certainty of result. I shall give briefly *my own routine*, premising that I do not regard it as essentially superior to any other that may be adopted.

After taking the history and getting at the symptoms I make an external and an internal examination of the eye, using in succession oblique illumination, direct illumination by transmitted light with the ophthalmoscope at ten inches, examination with the ophthalmoscope by the indirect method, and lastly examination with the ophthalmoscope by the direct method. From this I gather, whether the eye is healthy or not, the probable amount of vision, the presence of opacities or other defects interfering with sight, and approximately the state of the refraction. For the latter purpose I often add an offhand estimate with the shadow test.

Those who use the ophthalmometer should here make an examination with this instrument, and thus determine the amount of corneal astigmatism. The same thing may be roughly done with the Placido disk.

I then take the patient's vision, and begin the test with the trial case, proceeding in the way hereafter described to determine rapidly

the glass that corrects the manifest error. I at the same time test the patient's vision for near, and find the glass that suits him for reading. If, as in most instances, I wish to use a cycloplegic, I then instil homatropine, and, immediately, before the drug has time to act, make the muscle tests. Later, when the cycloplegic action of the homatropine is complete, or nearly so, I determine the refraction with skiascopy. Then I confirm or modify this result with the trial case, and finally check off these last results again by skiascopy.

In certain cases, as in children and in very nervous people, it saves time and is less tiresome to the patient to cut the subjective examination short, and to proceed at once to the objective examination with the shadow test, making this as carefully as possible. Using the correction thus found as a basis, I again make the test with the trial case, and now generally find that there is very little left to do in order to arrive at the final result, so that now I do not have to tax the patient's attention unduly nor make much demand upon his patience or judgment.

The Use of Cycloplegics. As we have seen, the myope may involuntarily exaggerate his myopia, and the hyperope wholly or partly conceal his hyperopia by using his accommodation. Patients whom we examine for glasses are so apt to do this that, if we wish to find out the precise refractive state of the eye, it often becomes necessary to abolish the accommodative effort altogether. This we do with a cycloplegic. The one ordinarily used is homatropine, a 2 per cent. solution of which instilled every five or ten minutes for five times produces in general complete paralysis of the accommodation in from an hour to an hour and a half from the time of the first instillation. The effect soon begins to wear off, and disappears entirely in from twenty-four to thirty-six hours. In instilling the homatropine it is best, as Jackson suggests, to have the patient throw the head back and look down, so that we may drop the solution directly on the upper part of the cornea. The eye in which the instillation is made should be held open until the patient voluntarily opens the other eye. This prevents his squeezing the drop out of his eye.

Homatropine is an irritant producing a moderate congestion of the eye, which, however, is transitory, and has no ill effects whatever.

Sometimes *scopolamine* in 0.1 per cent. solution is used, but in my experience it has no advantage over homatropine.

In cases where we wish to produce a very thorough and lasting effect—*i. e.*, in spasm of accommodation—atropine may be used in 1 per cent., or in particularly obstinate cases 1.5 per cent. solution. This is instilled night and morning for one or two days before the examination. The patient should be examined three hours after the last instillation. The paralysis of the accommodation in these cases lasts fully a week.

With the cycloplegic *near vision* for the hypermetrope and emmetrope is rendered impossible. Cycloplegics, besides abolishing accommodation, dilate the pupil. They hence render the vision

in ametropia worse, not only by preventing accommodative effort, but also by increasing the size of the diffusion images; and it is generally found that even with correction the vision under a cycloplegic is not so keen as with the pupil contracted. Moreover, the dilatation of the pupil, by letting in an excess of light, produces a troublesome *dazzling*. To obviate this, the patient may be directed to wear smoked glasses as long as the pupils remain dilated, this being especially necessary if he is exposed to bright sunlight.

The patient in whom atropine or homatropine has been instilled should be warned *not to try to use his eyes* for near work until the effect of the drug has completely worn off. Otherwise, by using his accommodation when still in a weakened state, he may strain the eye, and may even cause a condition of ciliary spasm. Hence a patient should not use his eyes for steady reading for thirty-six or, better still, forty-eight hours after the last instillation, if homatropine has been used; and not for eight or, still better, ten days if atropine has been used.

Hyperopes, who are so greatly dependent upon accommodation, are obviously very much affected by cycloplegics, their sight being rendered bad for distance and near. Myopes, on the other hand, for whom accommodation plays but little part in seeing, suffer but slight inconvenience.

Observers differ a good deal as to the *necessity of using cycloplegics* in determining refraction. In New York they are not so often employed as elsewhere, and many practitioners believe that they can determine refraction accurately without their aid. I do not share in this belief. I have seen a number of cases in which the refraction could not have been determined otherwise, and in which a serious error in the prescription would have resulted if no cycloplegic had been employed.

My experience, in fact, leads me to use a cyclopegic in all cases where I can when the patient is under forty-five years, and in some cases when he is between forty-five and fifty. It has been my experience that during the period from forty to forty-five a cycloplegic is especially important, as at this time patients are particularly apt to exert their accommodation excessively and hold on to it tenaciously. In these middle-aged patients I, of course, take care to exclude any suspicion of glaucoma, in which the instillation of a mydriatic would be disastrous. In my experience, homatropine properly applied is, in the vast majority of cases, fully as efficient and reliable as atropine. I find, too, that children yield readily to homatropine—quite as readily, indeed, as adults.

Every now and then we *fail to get complete relaxation* from the use of a cycloplegic. This is shown by the fact that the patient will take sometimes one glass, sometimes another, and that with the same glass his vision varies, so that, as he says, "the letters come and go," as he looks at the test card. Furthermore, the result of the objective examination with skiascopy or the ophthalmoscope will not agree with the glass found by the trial case.

8

When this happens, we give the cycloplegic more time to act, and in the meantime instil it several times again at short intervals. If this fails, we should use atropine in 1 to 1.5 per cent. solution several times daily for a series of days. But it is very rarely, indeed, that resort must be had to this expedient.

Keratometry. Placido Disk Determination of Corneal Curvature in Astigmatism by Keratometry. The cornea acts like a convex mirror, and will hence give a small, erect reflection of an object, such as a disk, placed in front of it. The more convex the cornea, the smaller this reflection is. What is true of the cornea as a whole, is true of each separate meridian of it. If, then, all the corneal meridians are equally convex, the reflection of a circular disk will also be circular; but if some meridians are more convex than others—that is, there is corneal astigmatism—the reflection will be oval, and the small diameter of the oval will lie in the meridian of greatest curvature or greatest refraction.

This principle is utilized in the application of the *Placido disk.* This is a white disk bearing a series of concentric black rings painted on it. (Fig. 2.) The observer, holding this before the patient's eye, and looking through the hole in the centre, sees the reflection of the disk in the cornea. If the reflection is circular, there is no corneal astigmatism; if it is oval, there is regular corneal astigmatism, the meridian of greatest curvature being in the short axis of the oval. If there is irregular corneal astigmatism, the reflection will be irregularly distorted, or will change its shape abruptly when shifted from one part of the cornea to the other.

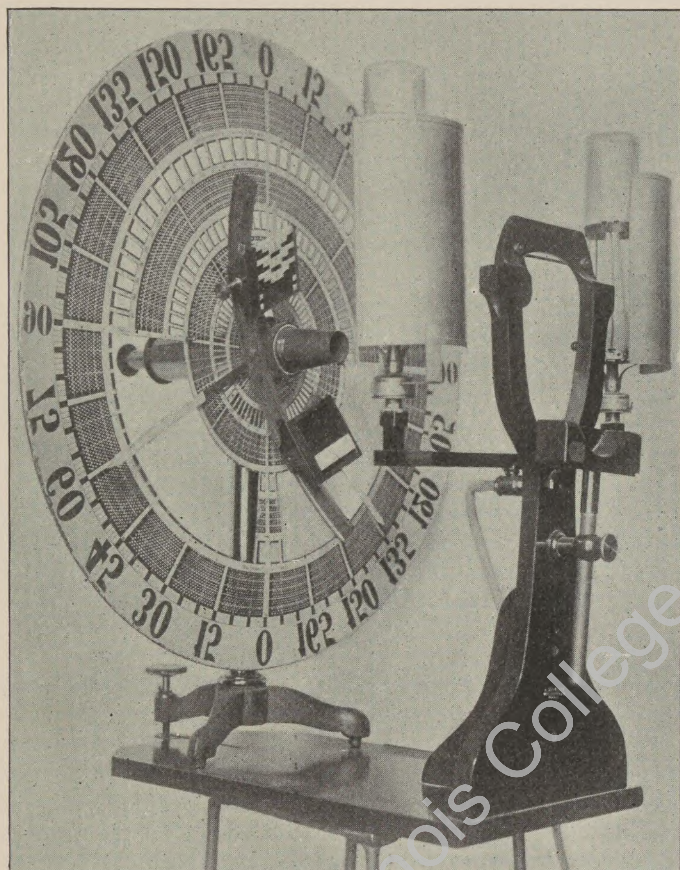
Since the corneal reflection of the Placido disk grows smaller as the curvature of the cornea becomes greater, and also becomes more oval as the corneal astigmatism increases, we may, by measuring this reflection accurately in its different diameters, calculate both the actual curvature of the cornea in all its meridians and the precise amount of its astigmatism. Both determinations are much more rapidly made with the ophthalmometer, which in principle is a specially modified Placido disk.

Ophthalmometer. The ordinary type of ophthalmometer is that devised by Javal and Schiötz. (Fig. 59.)

The ophthalmometer (Fig. 59) consists of a telescope, *A*, containing a double-refracting prism, a graduated disk, *B*, having a hole in its centre through which the telescope passes; and an arc, *C*, bearing two slides or mires, *D* and *E*. The arc *C* is attached to the telescope, which is so mounted as to turn freely in the central hole of the disk *B*, and as it turns carries *C* around with it. The hole is highly illuminated, usually by artificial light. The patient steadies his head on the chin-rest *F*, and looks into the large end of the telescope; the observer, observing at the patient's eye through the other end of the telescope, will see the cornea, and upon it the reflection of the disk and slides, but because of the double-refracting prism in the telescope will see two images of each slide. The two central images

of D and E, which are close together, alone are observed, the two outer ones being neglected. Each of these images is bisected by a dark line, and the telescope is revolved until the dark line of one image is approximately continuous with the dark line of the other. The line connecting the slides is then in one of the principal meridians of the cornea, that is, either the meridian of greatest or the meridian of least curvature. The mires are now slid to and fro along the arc

FIG. 59.



Javal-Schiötz's ophthalmometer.

C, until the images of D and E just touch. The telescope is then revolved through 90°, when the line connecting the slides will be in the other principal meridian of the cornea. If the images D and E now overlap or are separated, there is a corneal astigmatism of an amount proportional to their distance apart.

In the model of ophthalmometer shown in Fig. 59, the amount of overlapping of the images is measured directly. D is a parallelogram,

and E is a figure with a series of notches or steps. The instrument is so graduated that the number of steps by which E overlaps D represents the number of dioptres of corneal astigmatism.

In another model, when overlapping is present after the telescope has been rotated into the second position, the slides are separated until they just touch again. The amount by which the slides have been moved to accomplish this is read off on a scale behind the disk.

In both varieties of the ophthalmometer the *radius of curvature* of the cornea in any meridian can be determined. In the first model it is read off from the arc C, being shown by the distance between the slides when they are separated so far that their images on the cornea are just in contact. In the second form of ophthalmometer the radius of curvature of the meridian examined may be read off on a scale behind the disk. In both forms of ophthalmometer the index shows the situation of the meridian whose curvature is being measured, and in the case of the principal meridians it indicates the axis of the correcting cylinder, which must lie in either one meridian or the other.

The ophthalmometer does not show what kind of astigmatism—hyperopic, myopic, or mixed—is present. That is, it does not show what is the *absolute refraction* of the principal meridians, but only which of the two is the more refractive, and the difference in refraction between them.

Moreover, the ophthalmometer indicates simply the corneal astigmatism and its axis. It does not reveal either the amount or the direction of the total astigmatism, except in aphakia, where, the influence of the lens being removed, all the astigmatism is corneal. In any other case the result is only an approximation, although often a close approximation to the true findings.

OPHTHALMOSCOPY AS APPLIED TO THE DETERMINATION OF REFRACTION.

Direction of the Emergent Rays when the Fundus is Illuminated. When we throw light into the eye with an ophthalmoscopic mirror, and thus illuminate the various points of the fundus, these points themselves send out rays that diverge in every direction. On their way out these rays encounter the lens and cornea, which alter their course to a greater or less degree, according to the refractive power of the eye.

The eye being but a combination of lenses, the actual course taken by these emergent rays will be determined by the law that governs the direction of rays passing through lenses. This law, called the *law of conjugate foci*, may be thus stated: If a lens has such a strength that rays emanating from a point R, are brought together at a point N, then rays that emanate from the point N and travel back through

the lens will be *brought together at R*, and will form there a real inverted image of *N*. (Fig. 49.)

If the lens is of such a strength as to focus parallel rays at *N* (Fig. 43), then rays emanating from *N* will, after passing back through the lens, emerge *parallel*.

If the lens is of such a strength as to focus at *N* rays *D C*, that are converging to the point *R* (Fig. 50), the rays emanating from *N* will, after passing back through the lens, *diverge as if they came from R*, and will form at *R* an erect, virtual image of *N*.

To apply these principles to the eye, we may say that in emmetropia the rays that emanate from the illuminated fundus will emerge from the eye parallel to each other; in hyperopia they will diverge from the far point lying back of the eye; and in myopia they will converge toward the far point lying in front of the eye; and in either case will form at the far point an image of the portion of the fundus that is illuminated.

The behavior of emergent rays is the same, whether the eye is naturally emmetropic, hyperopic, or myopic, or whether it is made so by the addition of a convex or concave glass placed before the eye.

Determination of Refraction by Direct Illumination. If, when we stand off fifteen inches from the eye and then throw light into it, we see a clearly defined image of the optic disk and vessels, we know that this is the image formed by the eye itself at its far point, as shown in the preceding paragraphs, and that this image must be either between us and the eye (myopia of 6 to 7 D. at least) or behind the eye and close to it (hyperopia of 1 D. or more).

If, as we look into the eye, we move our head, this image of the fundus will move in the opposite direction in myopia and in the same direction in hyperopia. This is because we refer the movement to the plane of the pupil which lies behind the image in myopia and in front of it in hyperopia. It is the same experience that we get in a railroad train when, looking out of the window, we see objects in the foreground apparently running backward and objects in the far distance running forward.

Determination of Refraction by the Indirect Method. In using the indirect method, we should hold the object lens with its principal focus at the anterior focus of the eye, that is, about half an inch in front of the cornea. Hence a two-and-one-half-inch lens should be held three inches from the eye. When we do this, the size of the object seen in the fundus is not altered by the presence of axial hyperopia or myopia, and the distortion produced by astigmatism is relatively slight, so that the optic disk, for instance, appears normally round.

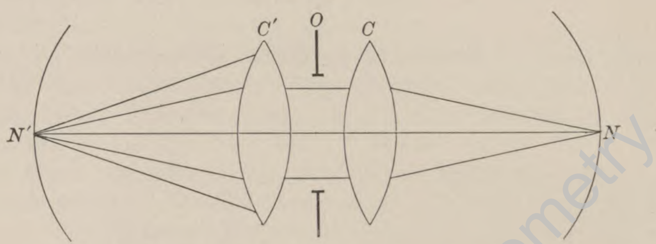
If, however, we carry the lens closer to the eye, then the apparent size of the disk diminishes in myopia and increases in hyperopia, while in emmetropia it remains the same. If we carry the glass away from the eye, the reverse change takes place, the optic disk becoming

apparently smaller in hyperopia and larger in myopia, while in emmetropia, as before, it remains unchanged. In marked hyperopia and myopia these changes take place quite rapidly.

The most striking effect of these alterations is seen in astigmatism. If we examine a patient with hyperopic or myopic astigmatism with the rule, the optic disk will look like a horizontal oval when our glass is close to the eye, round when we carry the glass back to the standard position, and a vertical oval if we withdraw the glass still farther. In astigmatism against the rule, the disk will be a vertical oval when the glass is close, and becomes a horizontal oval when the glass is withdrawn. In marked astigmatism these changes are very pronounced.

Determination of Refraction by the Direct Method. As we have seen, if we illuminate the fundus of an emmetropic eye, rays will emerge from it parallel to one another. If we ourselves are emmetropic, or make ourselves so with the proper glass, and relax our accommodation completely, we may, without additional aid, focus

FIG. 60.



Determination of refraction by the direct method. Rays emanating from the patient's retina, *N*, pass through his lens and cornea (*C*), and if the eye is emmetropic, or has been made so by glasses, emerge parallel to one another. The observer's eye receives these parallel rays, and, if he himself is emmetropic and not using his accommodation, will focus them upon his retina, *N'*. *C'*, observer's cornea and lens. *O*, ophthalmoscopic mirror perforated to allow the light to pass through.

these parallel emerging rays upon the retina, and will get a sharp image of the patient's fundus. (Fig. 60.) We will, in other words, using the ophthalmoscope, get a distinct image of the fundus if we look through the sight-hole simply.

If the patient is ametropic, we have now simply to bring before the sight-hole of the ophthalmoscope that lens which will correct his ametropia, and thus render him emmetropic. We shall thus, as before, see the fundus distinctly. We thus arrive at this rule: *the glass which we have to place before the ophthalmoscope in order to see the patient's fundus distinctly is the glass that corrects his ametropia.* Thus if we have to place a +4 D., he has a hyperopia of 4 D.; if we have to use a —3 D., he is myopic 3 D., etc.

In determining refraction by this method, we usually fix upon one of the fine vessels, preferably near the macula, or at least running from the temporal side of the disk, and ascertain the glass with which we can see it most distinctly. A more satisfactory test object still

is the fine granular markings of the fundus in the vicinity of the yellow spot.

In *astigmatism* we obviously cannot render the patient emmetropic by putting up any of the spherical glasses contained in the ophthalmoscope, consequently we cannot obtain a perfectly distinct view of the fundus. The most we can do is to render one of his principal meridians emmetropic. Suppose that he has astigmatism of 4 D. with the rule, and that his vertical meridian is hyperopic 1 D. As soon as we put up a +1 D. with the ophthalmoscope his vertical meridian will be emmetropic. With such a glass he would see horizontal lines distinctly. As he sees out of his eye so we see into it, getting the same kind of view of his fundus that he gets of the outside world. Hence with this +1 D. we shall see the horizontal vessels of the retina sharply, but the vertical vessels will be confused. If now we correct the other meridian by putting up a +5 D., we shall get the reverse effect, seeing the vertical vessels most distinctly and the horizontal ones very hazily.

This example shows us how we arrive at the rule that in astigmatism *the glass that gives us a clear view of the vessels running in the direction of one of the principal meridians measures the refraction of the meridian at right angles*, that is, of the other principal meridian.

The disk seen by the direct method will appear lengthened in the direction of the most refracting meridian.

To take another illustration: suppose that by putting up a +1 D. we notice the disk is elongated in the meridian of 80° and that we see the vessels in that meridian most sharply. With a —2 D. we see most sharply the vessels in the meridian of 170°. The refraction of the 80° meridian is —2 D., and of the 170° meridian is +1 D., and the correcting glass is +1 sph. ⌒ —3 cyl., ax. 170°.

The conditions for the successful *determination of refraction* by the direct method are as follows:

1. Our own refractive errors should be corrected by the proper glass and our accommodation completely relaxed. The ability to do this varies in different people, and I believe from what I have seen of the discrepant results obtained by different ophthalmologists, that their accommodation is seldom as completely under control as they believe it to be.

2. The patient's accommodation should be completely relaxed. It is generally supposed that he will relax perfectly if examined in a dark room without anything to fix upon. This is by no means always the case.

3. The ophthalmoscope containing the correcting glass should be held at the anterior focus of the patient's eye; this is a half inch in front of his cornea.

4. In the case of astigmatism we should be able to find vessels running in the direction of both principal meridians.

It is difficult to fulfil all these conditions with precision, and even with the aid of a cycloplegic which eliminates the error due to the

patient's accommodation, the findings should be characterized as approximate only.

The direct method is much used in estimating the relative refraction, and hence the *relative depth and prominence* of the different portions of the fundus. Thus if we see the bottom of an excavation in the disk with a —4 D. and the edge with a +2 D., we know that the depth is approximately 2 mm., corresponding to this difference of 6 D. So also an exudate or a detachment which we see distinctly with a +4 D., while the surrounding fundus requires only a +1 D. to make it visible, is 1 mm. high, corresponding to the difference of 3 D.

SKIASCOPY, OR THE SHADOW TEST.

Principle of the Test with the Concave Mirror. If we stand off at 1 m. from the patient and throw light into his eye with a concave mirror—for instance, the mirror of the ophthalmoscope—we shall see a light-red reflex filling the pupil. Then, if we turn the mirror slightly the light will gradually leave the pupil, and darkness will succeed it, until the pupil becomes entirely black. The direction in which the light moves in passing from the pupil depends upon where the far point of the eye is. If the far point is between the observer and the patient, the light will move off the pupil to the right when the mirror is turned to the right, and *vice versa;* that is, the light will move *with the mirror.* If the far point is not between the patient and the observer, that is, is either back of the head of the observer or of the head of the patient, the light will move *against the mirror*, or to the left when we turn the mirror to the right.

Instead of watching the movement of the light, it is usual to watch the movement of the dark area or shadow which follows it and moves with it, hence the term skiascopy, or shadow test, applied to this method of observation.

If then, standing at 1 m. we see the shadow move with the mirror, we know that the patient's far point is between us and him, and lies within 1 m. of the eye. He must then be myopic more than 1 D. If we slowly approach him until we reach a point when the shadow begins to move against the mirror, we know that at this distance we have just passed his far point, so that it is now just back of our head. The point at which this change of movement from with to against occurs is called the *point of reversal.* It obviously coincides with the patient's far point.

Instead of thus moving up to the patient to ascertain his point of reversal or far point directly, we usually stand at one distance, generally 1 m., and, by putting glasses on the eye we are examining, change its refraction until the same reversal takes place. When this has been effected, we have put his far point just back of us, that is, just beyond 1 m. With the next weakest glass we should have put his far point just at 1 m. and made him myopic 1 D. To make him

emmetropic, we should now give him —1 D. in addition, since any myope of 1 D. will be made an emmetrope by a concave glass of this strength.

If standing at 1 m. we get a movement of the shadow against the mirror, we know that the patient's far point is between us and him, that is, he is not myopic 1 D. or more. He may be hyperopic, emmetropic, or myopic less than 1 D. To determine his refraction and its precise character, we now add convex glasses, thereby increasing his refractive power, until we finally get a movement with the mirror. We have then just brought his far point down to within 1 m. and have made him myopic 1 D. As before, we make him emmetropic by giving him —1 D. in addition, or by subtracting 1 D. from the convex glass previously put on.

The actual practice of conducting the shadow test may be stated as follows:

We stand slightly more than 1 m. from the patient. If, using a concave mirror, we get a movement of the shadow *with* the mirror, we add concave glasses until the movement just goes against. The last glass (highest concave) with which the shadow still moves with the mirror is the *reversing glass.*

If the shadow movement is *against* the mirror to start with, we add convex glasses until it just begins to go with the mirror. The first glass (lowest convex) with which the shadow moves with the mirror is the *reversing glass.*

Add a —1 D. to the reversing glass, and the sum will be the *correcting glass* required.

Thus, if we get a movement with the mirror, and if by adding a —2 D. we get a movement against, while a —1.75 D. still gives a movement with, we know that the patient is myopic —1.75 + (—1) or —2.75 D. If, in another case, we get a movement against, which finally is converted into a movement with the mirror, by a +3 D., the patient's true correction is +3 + (—1) or +2 D. So also a movement against that would be just reversed with a +1 D. would indicate emmetropia (+1 D. + (—1 D.) = 0); and one reversed with a +0.25 D. would indicate a myopia of 0.75 D. (+0.25 —1 = —0.75).

Application of the Plane Mirror. Many use a plane mirror instead of a concave. This gives a brighter illumination and a better marked movement of the shadow, but, as I have repeatedly found, from the very largeness of the movement, the plane mirror is likely to give a confusing result in determining the reversal in astigmatism, and I am sure that I have gotten more accurate results with the concave mirror in these cases.

With a plane mirror the motion of the shadow is *just opposite that obtained with a concave mirror.* That is, when the far point is between us and the patient we get a movement against the mirror, and the rules above given should simply be reversed, the word "with" being substituted for "against," and *vice versa.*

Distance at Which Test is Made. We may stand at any distance in making the shadow test. We should then make a proportionate addition to the reversing glass in order to obtain the proper correction. If, for instance, we stand at $\frac{2}{3}$ m., the glass with which we obtain reversal at that distance puts the patient's far point at $\frac{2}{3}$ m., that is, makes him myopic 1.50 D. Hence we shall have to add —1.50 D. to the reversing glass to obtain the true correction. So, also, if we stood 2 m. we should have to make an addition of only —0.50 D. to our reversing glass, which in this case will make the patient myopic 0.50 D. In the majority of cases a distance of 1 m. is most practicable.

Testing the Result. To prove the result, we put the reversing glass before the eye, and, standing at 1 m. or a few inches beyond, note that the shadow goes with the mirror. We now approach a few inches. If our reversal is accurate, the shadow should now move against the mirror.

Character of the Reflex. If the ametropia is very high, the reflex is very dull—in fact, we scarcely see any light in the pupil. In proportion as we add correcting glasses and get nearer the reversal, the reflex becomes brighter, becoming very brilliant and white when the reversal is reached. Hence when we see a dull reflex not attributable to opacities of the media we add strong glasses at once (several D. at least); and if the reflex is still dull, change the glass for one which is 1 or 2 D. stronger. As soon as the reflex becomes bright we make slight changes (0.50 to 0.25 D.) in the glasses added.

The Test in Astigmatism. To determine astigmatism, the shadow test is made as follows: We put on glasses, + or —, until the movement in one meridian is reversed. When this occurs and the astigmatism is of any amount, the luminous reflex is converted into a well-defined *band of light* running precisely in the direction of the meridian that we have corrected. We then proceed to correct the meridian at right angles to this. We may do this by adding more spherical glasses until reversal is obtained in the second meridian also. When this is done, the band of light will be seen again; but it will now run in the direction of the second meridian, or at right angles to its former direction. The difference between the reversing glasses of the two meridians will give the astigmatism, and the direction of the band of light will give us its axis.

Thus, suppose that at 1 m. we get a movement against in all directions. With a +2 D. we see a band of light running at 75°. By careful addition of glasses we find that +2.25 D. just makes the shadow go with the mirror in this meridian. In every other direction the movement is still against. Making our mirror now move precisely in the axis of 165°, we find that when we put on a +4.50 D. the band of light lies in this axis, and when we put on a +5 D. the shadow just begins to go with the mirror. The reversing glass is then +2.25 D. in one meridian, and +5 D. in the other, and the astigmatism is the difference between the two, or +2.75 D. Adding

—1 D. for the distance of the point of reversal, we have as the true correcting glasses, +1.25 D. and +4 D., respectively, and the total glass correcting the error would be $+1.25 \bigcirc +2.75$ cyl. ax. 75°.

A more accurate way to correct astigmatism is, after we have obtained reversal in one meridian, *to leave on the reversing glass and add cylinders* with their axes in the line of the corrected meridian. Thus in the case stated we should, after finding that +2.25 reversed in the meridian of 75°, leave this glass on, and add + cylinders with their axes at 75° until reversal was obtained in the meridian of 165°. In this case, if our correction is accurate, we should get an even reversal not only in the meridian of 165°, but in all meridians alike, for our glass, if correct, abolishes the astigmatism and makes the patient simply myopic 1 D.

Our estimate of astigmatism by the shadow test will be faulty unless we *move the mirror strictly in one of the principal meridians.* If we swerve from this meridian, the shadow will make an oblique movement, apparently sliding off the line in which we swing the mirror. Hence if we do get an oblique movement, we should change the direction in which we move the mirror until we get it going right in the plane in which the shadow tends to move. In other words, we should so manage the mirror that when it moves the shadow will move precisely with or precisely against it, and not slide off the path.

This same tendency of the shadow to make an oblique or skew movement is noticed when we apply cylinders in making the test, and happen to have placed the cylinders somewhat out of axis. When this occurs, we should shift the axis of the cylinder until the oblique movement ceases.

In *irregular astigmatism* we get all sorts of irregular moving shadows, forming kaleidoscopic patterns on the pupil. Such a picture does not necessarily indicate an incorrigible condition, for we may by patience determine a more or less regularly moving shadow in addition, caused by a regular astigmatism which is susceptible of correction by glasses.

Central and Peripheral Shadows. In meridianal aberration, when the cornea has a different refraction in its centre and at its periphery, we get a double shadow. Thus, with the periphery emmetropic and the centre hyperopic 0.75 D., we will, with a +1 D. before the eye, see a shadow start from the top of the pupil and move down as we move our mirror down. At the same time we will notice a fine shadow start from some point in the lower half of the pupil and move upward, deepening as it proceeds.

Not until we have put on a +1.75 D. will this contrary movement of the central shadow be abolished, so that we get a uniform movement with the mirror clear across the pupil.

In pronounced cases of this sort we find two shadows developing near the centre of the pupil and going to meet each other, like the blades of a pair of scissors (scissor movement).

Usually in such cases the true refraction is that shown by the more interior shadow, and not by the peripheral one. Thus, in the case just cited, the correcting glass would probably be +0.75 D., corresponding to the hyperopia of the more central area of the pupil.

Skiascopy as a Confirmatory Test. One of the most useful applications of the shadow test is in confirming the glass found by subjective examination. Suppose, for instance, with the trial case we have found +1.50 D. sph. $\bigcirc$ +1.75 cyl. ax. 90°; we add to this glass +1 D., making +2.50 D. sph. $\bigcirc$ +1.75 cyl. ax. 90°. By so doing, we make the patient myopic 1 D. With this glass, when we use a concave mirror and stand at a little beyond 1 m. we should get a movement with the mirror in all meridians. Then, by going a few inches nearer the patient, we should get a movement against the mirror in all meridians. If this reversal does not take place for all meridians at the same instant, but occurs a few inches nearer the patient for one than for another, the astigmatism is not properly corrected, and we should change the strength of the cylinder accordingly until the reversal is perfectly even for all meridians alike. If, again, the axis of the cylinder is not correct, we will observe that the shadow makes a somewhat oblique movement, which is corrected when we set the cylinder at the proper axis. Finally, if the spherical glass is not correct, *e. g.*, if in the case cited it were +1.25 instead of +1.50, we should find that with our trial glass the reversal would take place rather nearer than 1 m.

The Use of Cycloplegics. In using the shadow test it is generally essential that the accommodation be relaxed with a cycloplegic, although in many cases we may get quite an accurate determination without this. Made with a cycloplegic, skiascopy is an extremely accurate test. If done with sufficient care, the refraction may be estimated up to within one-eighth of a dioptre.

SUBJECTIVE TESTS IN GENERAL.

Varieties of Subjective Tests. In all subjective tests of refraction we are dependent upon the information furnished by the patient himself as to what he sees. A number of such tests have been devised, some of which require the use of elaborate apparatus, called optometers, refractometers, etc. None of them has superseded the use of the trial case, which is not only the best subjective test, but which has to be resorted to in any event, whether other methods are used or not.

In general it may be said that these other subjective tests are of little service or else are superfluous.

Direct Determination of the Far Point in Myopia. In high myopia we may form a rough idea of the amount of the error by determining the farthest distance at which the patient sees fine print. If, for instance, he begins to read it at 3″ from the eye, we know

that his far point is 3″, or that his myopia is 13 D. This method, of course, is of very limited application, and gives only a rough approximation.

Astigmatic Clock-face. The astigmatic clock-face is frequently used as a subjective test. The clock-face, or fan, consists of lines or bundles of lines radiating from the centre of the dial, as shown in Fig. 61. If a man having hyperopic astigmatism with the rule looks at such a dial he will see the horizontal lines (those running from III to IX) most distinctly, as his vertical meridian is most nearly emmetropic. If he has myopic astigmatism with the rule, he will see the vertical lines running from XII to VI most distinctly; and if he has oblique astigmatism, the corresponding oblique meridian or the meridian at right angles to it will be clearest.

These differences come out most sharply when one of the principal meridians is emmetropic or has been made so by a glass. Hence many, in order to determine astigmatism, proceed as follows: They

FIG. 61.

Astigmatic clock-face.

FIG. 62.

Pray's astigmatic letters.

put on spherical (preferably convex) glasses until one line in the clock-face is perfectly sharp. Suppose this to be the vertical line, and that the spherical glass used is +2 D. Then, with this glass, the horizontal meridian must be emmetropic. Successive cylinders are now added with their axes horizontal until the clock-face appears uniformly sharp, so that the lines are perfectly defined. Suppose it takes a —1.25 cyl. 180° to do this. The combined sphere and cylinder +2 sph. ⊂ —1.25 cyl. ax. 180° or +0.75 sph. ⊂ +1.25 cyl. ax. 90° should then be the correcting glass.

If this method is adopted, the glass found should always be confirmed by a trial made with the test types.

Personally, I have found that patients differ so much in their estimates of the lines seen most distinctly and of the effect upon the distinctness produced by adding glasses, that I do not use the clock-face as a primary test, but rather use it at the end of the examination, to confirm the result obtained with the test types, and see if

the glass I have found with the latter makes the clock-face appear perfectly uniform.

A modification of the clock-face is *Pray's astigmatic letters*, which are block letters made up of horizontal, vertical, and various oblique lines. To the patient with astigmatism, some of these letters look quite black and some gray, according to the axis of the astigmatism.

Testing with the Trial Case and Test Cards. By far the best method of subjective examination, and one which should never be neglected when it can be used at all, is experimental testing with the trial case and test types. The trial case contains convex and concave spherical glasses from 0.25 D. or 0.12 D. to 20 D., and cylindrical glasses from 0.25 D. to 6, or in some cases, 8 D. It is always best to have these glasses in pairs.

The trial case contains also prisms, a blinder for covering one eye when the other is being examined, red and other colored glasses, disks containing stenopæic slits or perforations, etc.

The trial frame for holding the glasses used in testing should be strong and steady. It has two, or, in some frames, three cells on each side, into which the glasses are slipped. In a good frame these cells may be readily moved in and out from the nose, forward or back, toward the eye, and up or down. We are thus enabled to centre accurately the glasses placed before the eyes; and we should take care to do this in every case we are testing.

General Rules. In the examination with the trial case the following general rules should be borne in mind:

RULE I. *One eye should be tried at a time*, the other being covered, not closed. The vision of the eye tested should then be taken.

RULE II. *The strength of the glass we try* before the eye should be selected according to the patient's vision at the time. This is true both of the glass we begin with in order to get the first approximate correction, and of the successive glasses we add to this approximate correction, in order to get nearer and nearer to the true result.

Thus, if we have a patient with vision of 20/200 or less and we feel pretty sure from the objective examination that the poor sight is due to his refractive state alone, we would begin at once with a spherical glass of from 3 to 5 D. (+ or — as the case required). It would be of no use to try a much weaker spherical or any cylinder, as a patient with this vision would not appreciate the difference made by such a glass. If the vision were thereby increased to 20/70 or 20/20, we would add a spherical glass of 1 to 2 D. When the vision had become 20/40 or 20/50 we would add 0.75 sph. (or if applying cylinders a 1.00 D.) to the correcting glass already in. With vision of 20/30+ or 20/30— we would add 0.50 D. (sphere or cylinder). Finally, when the vision had become 20/20 we may add a glass of 0.25 D., as then the patient may be able to notice the very slight difference that a glass of this strength produces.

This rule no longer holds good when the patient has poor vision due to opacities in the media, or in the retina or nerve. Then

a weaker glass than that indicated by this rule will often produce an appreciable alteration in sight. But in such cases it is best to proceed according to

Rule III. In nervous and fidgety persons, or in children when they get tired and inattentive, or in any case when, from defect in the eye itself or from lack of mental training, the patient cannot tell what he sees, we should *drop the testing with the trial case* altogether, instil homatropine and determine the refraction carefully by skiascopy. When we have made as careful a determination as possible by this method, we may then, with the glass thus found, resort to the trial case to confirm our result.

Rule IV. *In examination without a cycloplegic* we select as a measure of the refraction the highest + and the lowest — glass that gives the patient the best vision. It is evident that a man having full use of his accommodation and seeing equally well with a +2 D. and a +2.25 D. glass, should have at least +2.25 D. hyperopia, for if a +2 D. really made him emmetropic, the addition of even +0.25 D. would blur his sight. So also, if a patient can see as well with a +0.75 D. as without it, he must have at least 0.75 D. hyperopia, the latter being measured by the highest + glass that he *accepts*. Again, if the patient were myopic 1.50 D. he would see well not only with a —1.50 D., but by using his accommodation, also with a —1.75 D. or a —2 D., which would over-correct his myopia; hence the lowest of the three glasses would be the real measure of his refraction.

Rule V. When, on the contrary, we examine *with a cycloplegic*, we select as a measure of the refraction the lowest + and the highest — glass that gives the patient the best vision.

Rule VI. As we have seen, the strength of a concave glass is diminished and that of a convex glass is increased when the glass is carried away from the eye. For this reason when testing refraction with lenses in the trial frame, we should be sure that they are *at the same distance from the eyes* that the patient's glasses will be when he wears them; otherwise we will make an error in our estimate, an error which may be of sensible amount. If, for instance, the trial frame stands out too far from the face, our estimate of a — glass will be too strong and of a + glass too weak. In the case of strong glasses the error will amount to a whole dioptre.

Rule VII. If, in the course of the investigation, it becomes apparent that *the vision cannot be readily brought to normal by any glass* tried, it is best to re-examine the eye with oblique illumination and with the ophthalmoscope, to see if any opacity in the media, disease of the fundus, or marked irregular astigmatism may not be present, causing an incorrigible defect. Very often the use of a mydriatic is necessary to reveal such conditions, especially in the case of lesions in the yellow spot. Examination of the field of vision is also often very helpful, as it may reveal loss of central vision (due perhaps to tobacco amblyopia, etc.) or a marked and increasing contraction of the field, indicative of a neurasthenic state that causes

the amblyopia. We should also be on the lookout for the amblyopia of an eye that is squinting or once was subject to squint.

Routine of Procedure. My own procedure in using the trial case is as follows: I first *apply the approximate correction,* determined by the cursory examination made with skiascopy or with the ophthalmoscope. This correction would, in general, be a spherical glass, *A,* and a cylinder, *B.*

Second, I add to this correction a series of glasses selected according to what I call "*the round of the trial case.*" That is, I add one after another quickly:

1. A convex sphere.
2. A convex cylinder with its axis in the axis of *B.*
3. The same cylinder with its axis at right angles to *B.*
4. A concave cylinder with its axis in the axis of *B.*
5. The same cylinder with its axis at right angles to *B.*
6. A concave sphere.

These additions form a series of combinations which represent practically all changes that can be made in the sphero-cylinder *A B.* Thus, if *A* and *B* are both convex, the additions made will:

1. Add to *A* and leave *B* alone.
2. Add to *B* and leave *A* alone.
3. Diminish *B* and add to *A.*
4. Diminish *B* and leave *A* alone.
5. Increase *B* and diminish *A.*
6. Diminish *A* and leave *B* alone.

For example, if $+2.50 \frown +1.25$ cyl. 90° were the glass originally in the frame and a glass of 0.50 D. were added in making the "round," the successive additions made and the combinations resulting therefrom would be as follows:

Additions.	*Resulting combination.*
+ 0.50 sph.	+ 3.00 + 1.25 cyl. 90°
+ 0.50 cyl. 90°	+ 2.50 + 1.75 cyl. 90
+ 0.50 cyl. 180	+ 3.00 + 0.75 cyl. 90
— 0.50 cyl. 90	+ 2.50 + 0.75 cyl. 90
— 0.50 cyl. 180	+ 2.00 + 1.75 cyl. 90
— 0.50 sph.	+ 2.00 + 1.25 cyl. 90

The strength of the successive glasses added in making this round will be governed by Rule II.

Third, as soon as by making the round I reach a combination that improves the sight, I *substitute this new combination* for the old one. With this new combination as the approximate correction I start again on the round of the trial case, not usually, however, from the beginning, but adding a glass similar to the one that gave improvement before. Thus if I before got improvement by crossing with a — cylinder, I keep on crossing — cylinders until I fail to improve; then I try the next manœuvre of the round.

By making successive substitutions I soon get a glass which is not improved by any additions (+ or — sphere or + or — cylinder).

Then I make *minute variations in the axis of the cylinder* and see if this is just right.

Often the patient cannot indicate the axis with precision, saying, for instance, that he sees equally well with the cylinder at either 60° or 70°. In such a case I turn the cylinder up (toward 90°) until the vision is evidently blurred, then bring it back until the sight begins to clear again, and then take the reading. Suppose this to be 80°. I now rotate the cylinder down (toward 0°) until again the sight blurs, then once more bring the cylinder back until the sight clears up again. Suppose this to be at 50°. Then the axis must lie between 50° and 80°.

Working between these limits, I try again to find at just what point below 80° and at just what point above 50° the sight reaches its greatest clearness. Suppose I can thus narrow the limits down to 75° and 65°. Then the axis of the cylinder in all probability lies midway between these limits, or at 70°.

When this point is settled satisfactorily I may assume that my correction is perfect, but, to be sure of it, I usually *prove it by the shadow test* in the way already described.

The changes and successive substitutions in this round of the trial case are made with great rapidity, indeed, after a while, almost mechanically, and thus in a very short time the proper glass can be determined, and, in particular, we can be sure that no other glass than the one we have fixed upon will answer as well.

In order to determine whether the *vision with the pupil dilated* by a cycloplegic represents what the vision will be when the pupil contracts to its normal size, I slip a disk with a 4 mm. aperture in it over the correcting glass in the trial frame. I thus, in effect, reduce the pupil to the normal size and eliminate the disturbing effect of the rays passing through the periphery of the pupil, which often are not refracted like those passing through the centre. In most cases this disk will improve the sight, and, in particular, will make the test letters look sharper and blacker. If this is the case, I feel confident that the correction is the proper one, and will, after any necessary reduction in the spherical component, be accepted when the effect of the cycloplegic has subsided.

It should be noted that the test at 20 feet does not always give the best results for *far distance*. It is well, therefore, to try the glass that we have found by having the patient look out of the window into the far distance and then to observe whether a — 0.25 D. added does not sharpen the sight materially, defining, for instance, the lines of bricks and mortar, etc., in distant houses better. If it does, the corresponding reduction should be made in the glass determined for distance.

After I have determined the correcting glass for each eye separately, I often find it an advantage, especially in testing hyperopes without a cycloplegic, to put the *correction for each eye in the frame*, and have the patient look at the test cards with both eyes at once. It may

then turn out that he will take some addition to the correction in one eye or both which he did not take before.

The test for near is made simply by putting on each eye its proper correction and then making the patient use both eyes, and find what additional glass he needs for his reading or working distance—*i. e.*, we make the patient essentially an emmetrope by glasses, then conduct the examination in the same way as for emmetropes. (See Chapter II.) This examination, of course, should be made either before instilling the cyclopegic or else some days after the effect of the latter has subsided.

An example will indicate more clearly the method of procedure. A patient not under a mydriatic has vision of 20/20. I put on him +0.50, then +0.75, then +1.00 D., which he accepts; but he rejects a +1.25 D., that blurring his sight a little. Leaving the +1.00 sph. in, I add +0.50 cyl. ax. first at 90°, then at 180°. Both are rejected, but the former gives less blurring. I then add —0.50 cyl. 180°, which converts the +1.00 into +0.50 ⊂ +0.50 cyl. ax. 90°. This sharpens the sight considerably, and I immediately substitute +0.50 ⊂ +0.50 cyl. ax. 90° for the +1.00. As the patient shows astigmatism with the rule, I follow the lead that he indicates by now adding successively +0.50 cyl. ax. 90°, and —0.50 cyl. ax. 180°. With neither is the sight better, nor, in fact, is it as good as without the glass. Now making the "round" with a +0.25 D. sph. and cylinder, I find that the sphere improves more than the cylinder, indicating a correction of +0.75 sph. ⊂ +0.50 cyl. ax. 90°. The patient accepts no further change. I now rotate the cylinder and find that the sharpest vision and also the completest equalization of lines of the astigmatic clock-face are obtained with the cylinder at 80°. The final correction, therefore, is +0.75 sph. ⊂ +0.50 cyl. ax. 80°.

The other eye tested in the same way shows +0.50 sph. ⊂ +0.75 cyl. ax. 100°. With both eyes together he accepts an addition of +0.50 sph., and for reading, takes a further addition of +0.75 sph. His distance glass, therefore, would be R. +1.25 sph. ⊂ +0.50 cyl. ax. 80°; L. +1.00 sph. ⊂ +0.75 cyl. ax. 100°. His reading glasses would be R. +2.00 sph. ⊂ +0.50 cyl. ax. 80°; L. +1.75 sph. ⊂ +0.75 cyl. ax. 100°

Take another instance: A patient under homatropine has indicated by the ophthalmoscope and the shadow test a glass of about +1.75 sph. ⊂ +1.00 cyl. 75°. With this he gets 20/40 vision. I add in succession +0.50 sph., +0.50 cyl. 75°, +0.50 cyl. 160°, —0.50 cyl. 75°, —0.50 cyl. 165°, and —0.50 sph. I find that, of all these combinations, —0.50 cyl. 165° improves the most. I substitute the corresponding combination, +1.25 sph. ⊂ +1.50 cyl. ax. 75°, and now get 20/30+ vision. I again add —0.50 cyl. ax. 165°, but fail to improve; then +0.50 cyl. 75°, with the same result. —0.50 sph., however, seems a little better, and when I try the "round" with 0.25 D. glasses I get improvement with —0.25 sph. Making the "round" again, no glass added helps. I change the axis of the cylinder

and fail to get any certain result, the patient's answers being rather vague. The patient, therefore, seems to have +1.00 sph. ⊂ +1.50 cyl. 75° To prove it, I put in the trial frame a +2.00 sph. ⊂ +1.50 cyl. ax. 75° and apply the shadow test at 1 m. I find that the shadow moves with the mirror in all directions, but there is a slight oblique movement which is corrected by rotating the cylinder until it stands at 60°. I then find that I get complete reversal in the meridian of 60° when I stand just within 1 m., but do not get reversal in the meridian of 150° until I go back some distance beyond 1 m. The cylinder then needs strengthening. Substituting +1.75 cyl. ax. 60° for the +1.50 cyl. already in, I now find that I get complete even reversal in all meridians alike at 1 m. The correction is, therefore, +1.00 sph. ⊂ +1.75 cyl. 60°. With this we find the patient now gets 20/20+, and the astigmatic clock-face is perfectly even. He gets even sharper sight when I put up a diaphragm with a 4 mm. aperture, which, in effect, restores his pupil to the natural size. But when he looks out of a window he sees far distant objects better with —0.25 added. Hence his full correction for distance is finally determined as +0.75 sph. ⊂ +1.75 cyl. 60°.

Rules for the Prescription of Glasses. It being supposed that we have found a patient's total refractive error, we now have to determine what glass to prescribe for him. This depends upon so many factors that no rule can be laid down which will cover all cases. The following are the general principles that I follow:

1. I correct all the *astigmatism* that the patient has, unless it is over 6 D., in which case he is sometimes more comfortable and gets quite as good vision with the astigmatism slightly under-corrected. I do not, however, hesitate to prescribe cylinders of more than 6 D., when these give appreciably better sight than glasses of less strength

Astigmatism of only 0.25 or 0.50 D. I do not prescribe for, unless the symptoms (asthenopia, blurring of sight, etc.) seem particularly to call for the correction, or unless the patient has to use his eyes for very close and continuous near work, or finally, unless the patient is going to use a glass anyhow, in which case I regularly add the cylindrical correction that he requires, however small.

2. I correct the full amount of *myopia*, and, wherever I can, have the patient use the same correction for distance and near. If he is much beyond the age of forty years, this is, of course, impossible; and even below that age we may have to give the myope different glasses for reading and distance, particularly if he has not used concave glasses before for near work. But in myopes under forty years of age I make the attempt at any rate to get them to use their full correcting glass for all purposes, and I find that I generally succeed if the patients are at all faithful in following my instructions.

I consider it particularly important to correct myopia, whether of low or of high degree, in children, and to make them wear their glasses constantly. I am very strongly of the belief that this course

tends more than anything else to retard the further development of the myopia.

It is also of very great importance to correct fully the myopia, and to insist upon the patient's using his glass for all purposes when he has a convergence-insufficiency. The evidences of this will be an exophoria which is much more marked for near than for distance, and a tendency to recession of the near point of convergence, indicating that the ability to maintain binocular fixation at near points is failing. In such a case the use of fully correcting concave glasses for both distance and near, by stimulating the accommodative power, may prevent the development of a divergent squint.

Myopia of very high degree may have to be under-corrected on account of the annoyance that the very strong glasses often produce. But I believe that we may generally prescribe glasses up to 18 D., and in certain cases may give even stronger glasses with advantage.

3. I correct all the absolute and all the manifest *hyperopia*.

The latent hyperopia I under-correct according to:

a. The *age* of the patient. The older he is the less, in general, we can leave uncorrected. No absolute rule can be laid down, but it may be stated that in children under twelve years of age, from 1 to 2 D. of latent hyperopia may often be disregarded; from twelve to twenty-five years of age, 0.75 to 1.25 D.; and from twenty-five to forty years of age, 0.50 to 0.75 D.

b. The *conditions under which he works.* If he has to use his eyes excessively, especially at some trying kind of near work, and particularly if by artificial light, I should give him a stronger glass than if he were using his eyes mainly for distance and by daylight.

c. His *symptoms.* A patient with asthenopia, headache, and other evidences of eyestrain, will require a fuller correction of his hyperopia than one who has no such symptoms. So, also, one suffering from accommodative weakness, due either to neurasthenia or to the effects of recent disease, will require the correction of nearly or quite all of his hyperopia, even if of low degree.

d. The *muscular conditions.* A patient with esophoria, and, still more, one with an actual convergent squint, due to convergence-excess, should wear constantly the full correction of his hyperopia or within 0.25 D. of it. Experience has shown that it is only by the persistent and long-continued wearing of the full correction that the best results are obtained in these cases.

Per contra, a patient with marked exophoria, and particularly an exophoria due to convergence-insufficiency, often does better if his hyperopia is moderately under-corrected.

A simple hyperopia of 0.25 to 0.75 D., or, in children, of even 1.00 D., may in most cases be left uncorrected unless it is producing blurring of sight, asthenopia, or a convergence-excess.

In *anisometropia,* whenever the sight in both of the eyes can be brought up to anything like the normal, I try to either give the full

correction in both, or else reduce the full correction by an equal amount in both eyes, and I tell the patient that he should wear the glass steadily, that he may expect to have some trouble with it for the first week, and, possibly, for the first two weeks, but that he should persevere with it, nevertheless. The indication for thus correcting anisometropia I regard as particularly important when, as often happens, there is a marked tendency to deviation of the eyes (insufficiency or squint).

By attending to the considerations above presented, it is often possible to prescribe the proper glass at once from the results of the first examination, made with a cycloplegic. In some cases, however, particularly when the results of examination before homatropine and under homatropine are very discrepant, and particularly, also, when there is doubt as to the proper reading-glass to be given, it is safer to have the patient report for *re-examination a day or two after the effects of the cycloplegic have worn off*. When he does so, I put on each eye the full sphero-cylindrical correction found under the cycloplegic, and, having the patient use both eyes, find what he can see. If he does not get full vision, I change the spherical glasses (but, if possible, do not change the cylinders) until his sight is brought to the normal. This gives me his *manifest* correction. The difference between this and the glass found under the cycloplegic is his latent hyperopia. Then the glass I prescribe for him will be the manifest correction plus a spherical glass whose strength represents that portion of the latent hyperopia which, I think, ought to be corrected, taking into account the patient's age, the kind of work that he has to do, the amount of his asthenopia, etc.

If the patient has a convergent squint, I pay no attention to manifest correction, but, as before stated, give the full, or practically the full correction, no matter whether the patient has poor sight with it, or not, and direct him to use it constantly.

Whether a glass shall be *worn constantly or not* depends upon the amount and kind of refractive trouble present and upon the symptoms. Persons with astigmatism of 1 D. or over, persons with myopia, whether low, moderate, or high, and persons with a hyperopia that is sufficiently high to occasion asthenopia or other evidences of eyestrain, should wear their glasses all the time. And in most other cases, if glasses are to be worn at all, it is better for the patient to use them constantly, provided, of course, they are not prescribed simply for presbyopia. When, however, a convex spherical or cylindrical glass is of slight amount and is prescribed for an asthenopia occurring solely in near work, the patient may be permitted to do without the glass for distance. Yet even in this case it is best to direct the patient to wear the glass continuously for a time, as by so doing he will accustom himself to it much more quickly. I usually tell such patients that they should wear their glasses all the time for three or four weeks, and that then they may discard them for distance if they choose. Often at the end of the time set they will of their

own volition continue to wear the glasses constantly—which will, on the whole, be a good thing.

A constant wearing of the correcting glass is particularly indicated when there is (*a*) a convergence-insufficiency combined with myopia, or a convergence-excess combined with hyperopia, or (*b*) a spasm of accommodation, or (*c*) a beginning myopia in childhood, or (*d*) a pronounced asthenopia with its attendant symptoms of eyeache and headache, or (*e*) neurasthenia with eye symptoms and sometimes, also, when no eye symptoms are present.

Difficulties Encountered in Wearing Glasses. Many people experience difficulty when they put on glasses for the first time. Generally speaking, these difficulties vanish after the glasses are used for a few days, particularly if they are worn constantly. Hence, if a patient does come back two or three days after getting his glasses and complains that he cannot see well or comfortably with them, he should be told to put them on and wear them steadily for at least a week, and that he should not be discouraged if he does not become perfectly accustomed to them before the week is over. In fact, we should, as a rule, not think of changing a glass simply on account of the difficulty experienced in using it until constant use for at least three weeks has proved it unsuitable. In the great majority of cases it will be found at the end of this period that whatever trouble the glasses occasioned at the outset will have entirely disappeared, provided proper care was taken in prescribing the glasses in the first place.

The difficulties experienced in using glasses may arise from changes they produce in the *size* of objects. Myopes often complain that their glasses make objects, especially objects close to them, look too small, so that they find difficulty in reading, sewing, or doing any fine close work with the correction prescribed for distance. Usually this difficulty passes away after a while, and, in the expectation that this will take place, we should always encourage the myope to keep on with his glasses for a week or two at least, in order to see if he will not gradually get accustomed to them for all purposes. If, as exceptionally happens, he cannot get used to them, then we shall have to give him a weaker glass for near work. How much weaker must be determined by experiment; but in any case, if he is under forty years of age, we try to get his reading-glass as near to his distance-glass as possible.

Hyperopes sometimes complain that their glasses make near objects look too large and coarse. This difficulty is probably always remedied by more prolonged use of the glasses.

Glasses also cause trouble by altering the *shape* of objects looked at. This is particularly the case with cylinders, which often make a square appear oblong, or, more frequently still, trapezium-shaped (narrower at the top than at the bottom), and make horizontal surfaces appear sloping. Prismatic glasses have the same effect, and so do spherical glasses when the wearer looks obliquely through them or sees through their edges. This distortion produced by glasses is

often quite annoying, but usually passes off soon (within a week or two in most cases). Very seldom we have to obviate it by reducing the strength of the glasses.

Another disagreeable effect produced by glasses is an alteration in the *distinctness* of objects looked at. Hyperopes often complain of the blurring produced by their convex glasses when these over-correct, even by a very little, their manifest hyperopia. Sometimes even very prolonged use of the glasses does not obviate this, as they hold on very tenaciously to their accommodation, and so do not allow any further amount of their latent hyperopia to become manifest. We should in such a case make the patient use his glass steadily for three or four weeks. If at the end of this time there were signs that the excessive accommodation was giving way, or, in any case, if the blurring was not very annoying, we should continue the glasses for a further period of three weeks. If, however, the blurring continues, and particularly if it is causing the patient much annoyance, we must then weaken his glass somewhat, telling him that later on he will need to have it made stronger again. In particularly obstinate cases, where the proper glass causes persistent blurring, and where the weaker glass that would give better sight is not sufficiently strong to relieve the asthenopia, we will have to use a course of atropine (1 to 1.5 per cent solution instilled three times a day, for a week or two).

Quite the same blurring is found in myopes with a spasm of accommodation. But here we should by no means yield to the patient's desire for a stronger concave glass, but insist upon his wearing the proper correction as found under a cycloplegic. If the accommodation remains still in a state of spasm, we should, as in the hyperopes, use atropine, instilled three times a day, for two weeks.

Blurring is often noticed with convex glasses that have been prescribed for near work, whether in hyperopes or presbyopes. If this persists, we should alter the strength of the glass accordingly, being careful to test the patient as nearly as may be under the same conditions as those under which he works.

Myopes and astigmatics sometimes complain that their vision is *too sharp* with their glasses. They miss the mitigating haze that formerly invested all objects that they saw. They soon, however, become used to the new conditions, and learn to enjoy the increased definition and clearness of everything they look at.

Another way still in which glasses cause trouble is by altering the *muscular relations* of the eye. A convex glass, by doing away with the necessity for using the accommodation, may cause a temporary convergence-insufficiency, which produces a tendency to diplopia and consequent blurring of sight at near points. If this does not, as is usually the case, soon disappear with the continued use of the glasses, we may have to exercise the convergence with prisms, base out, or in some cases even have to diminish the strength of the convex glass. The prescription of prisms, base in, for constant wear is not advisable in these cases, since, although affording temporary relief to the

symptoms, it is apt to cause a progressive increase in the deviation.

So, also, a concave glass, by compelling the use of the accommodation, which before was not in use, may produce a temporary convergence-excess, and this may likewise cause slight diplopia and blurring for near vision. This, if persistent, may be remedied by practising the divergence at near points (one foot or less) with prisms of 15° or 16°, base in. The prescription of prisms, base out, for constant wear is not advisable in these cases.

If a *muscular deviation is already present*, the troubles to which it gives rise may be accentuated by glasses. This is particularly the case if the deviation is such as to produce diplopia. This diplopia may not occasion trouble so long as one or both of the double images are faint, so that one of them can be readily ignored. But when the glass makes both images distinct, they can no longer be neglected, and both obtrude themselves disagreeably upon the patient's notice, causing a sense of confusion and uncertainty that amounts to absolute distress.

Lastly, a disturbance in muscular relations may be produced by the *prismatic effect* of the glasses. All glasses act as prisms if their wearer looks through their edges instead of their centres or if they are decentred. If he looks nearly through the centre of one glass and through the edge of the other, or, if the glasses are of different strengths, and he looks through the edges of both, he will, in effect, have a different prism before one eye than before the other, and will hence tend to see double. If he actually sees double, his sight will be confused; if he overcomes the diplopia by muscular effort, he is likely to suffer from asthenopia. This often happens with strong glasses which, owing to their weight, are apt to sag and get out of adjustment.

The remedy for this is to see that the glasses are very carefully centred, or, if decentred, are so disposed as to produce an equal prismatic effect.

These various difficulties due to glasses are, of course, more apt to be produced with *strong glasses* than with weak ones; yet it has been my experience that very strong spherical glasses, and in particular, very strong cylinders are borne without much difficulty—in fact, quite as well as those that are much weaker.

Some of the greatest difficulties are experienced in *anisometropia.* Yet even here, if persistent efforts are made for a couple of weeks to use the glasses properly correcting both eyes, the difficulties will, in most cases, ultimately disappear, and the sight will be much better and more satisfactory with the glasses than without them.

The Adjustment of Glasses. It is important, as we have just seen, that glasses, especially if at all strong, should be accurately centred—*i. e.*, their optical centres should lie opposite the centres of the pupils. Moreover, the nose-piece should be so adjusted as

not to allow the glasses to oscillate or become tilted. Otherwise, the glasses may produce a prismatic or a cylindrical effect which was not intended.

The frames should, in general, be so adjusted that the glasses will stand at the anterior focus of the eye—*i. e.*, about one-half inch in front of the cornea.

Glasses intended mainly or exclusively for reading should be dropped some 5 or 6 mm. and tipped forward about 15°, so that when the gaze is directed down, as it is in reading, the line of sight may strike the glass at right angles and pass directly through its centre. Moreover, the centre of each glass should be carried 3 mm. in toward the nose; otherwise the eyes when converged, as they are in reading, will look through the inner edges and not the centres of the glasses.

These are points that the optician who makes the glasses usually attends to carefully. Yet it is always well for us to verify this adjustment personally, and satisfy ourselves that the glass is just of the strength we ordered, that it fits properly, and that it is steady. Otherwise, we may, at times, be blamed for troubles produced by the glasses, but which are due to faulty fitting, and not to any error in our prescription.

Sometimes our patients cannot go in person to the opticians to be fitted. Then we have to send along with the prescription, measurements and directions showing how the glasses are to be made up.

For spectacles these measurements and directions are as follows:

Size of glass.

Distance between centres of pupils (interpupillary or pupillary distance).

Style of bridge (whether C bridge, saddle-bridge, or snake-bridge).

Height of bridge—*i. e.*, height of top of bridge above the line connecting the centres of the pupils.

Width of bridge at base.

Distance of top of bridge in front of or behind the frame (in case the glasses are to be set in or out from the eyes).

Style of temples (whether straight or hooks).

Length of temples.

Material of frame and style (whether rimless or not).

In the case of eyeglasses there should be indicated:

Size of glass.

Interpupillary distance.

Style of frame. (This in general has to be done by reference to a catalogue or to certain standard forms.)

Width of nose-clip at top and bottom.

Distance by which glasses are to be set in or out from the general plane of the frame.

Material and style of frame (whether rimless or not).

Most of the trial frames have scales marked upon them, by the

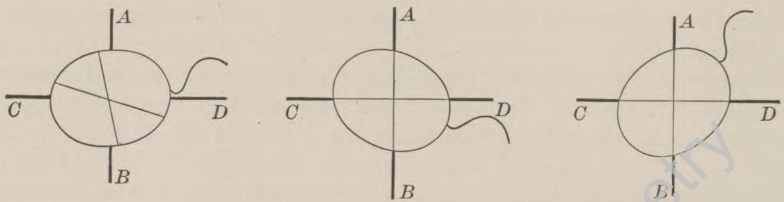
aid of which we are enabled to make the more important of the above-mentioned measurements.

Analysis of Glasses. In office work we are often called upon to examine the glasses a patient is wearing, and find out what they are. This is best done in the following way:

We hold the glass to be examined six or seven inches in front of our eyes and look through it at a right-angled cross (*e. g.*, that formed by two intersecting window sashes) on the other side of the room. We then rotate the glass about its centre, and watch the effect produced on the arms of the cross.

If, as we rotate the glass, the arms of the cross appear neither displaced nor distorted, but remain quite unbroken, we know that the glass contains neither a prism nor a cylinder. If in this case we move the glass from side to side and up and down, and no movement of the portion of the cross seen through the glass takes place, the glass is *plane*. If movement does take place, the glass is *spherical*, being

FIG. 63.



I II III

Distortion produced by a cylinder. Determination of the axis of a cylinder. A right-angled cross, *A B C D*, is seen through a glass containing a cylinder. If (I) the axis of the cylinder does not coincide with either *A B* or *C D*, the cross will appear twisted, so that the arms no longer make a right angle. The cross, however, is not displaced as a whole either to one side or the other. If now the glass is rotated until the axis of the cylinder coincides with one arm of the cross—*e. g.*, *A B* (II)—the cross will appear right-angled and unbroken. The same thing will happen if the glass is rotated 90° more (III), so that the axis of the cylinder coincides with *C D*.

concave if the cross moves in the same direction as that in which we move the glass, and convex if the cross moves the opposite way. In either case, we neutralize with glasses of opposite sign, putting convex glasses over concave, and *vice versa*, until all movement of the cross is abolished. The strength of the glass that precisely neutralizes the movement will equal the strength of the glass examined.

If the glass contains a *cylinder*, the two arms of the cross will bend toward each other as we rotate the glass, so as to form an oblique, instead of a right angle. The cross will, therefore, be distorted, but will not be laterally displaced.

If we keep on rotating the glass, we shall find the distortion first increasing, then diminishing, until finally a position is reached where there is no distortion—*i. e.*, where both horizontal and vertical arms are unbroken. The same will be the case if we rotate the glass 90°, so that the vertical arm occupies that meridian of the glass which

the horizontal arm occupied before. In either position of the glass the axis of the cylinder will lie precisely in the line where either the vertical or the horizontal arm of the cross traverses the glass.

Now, holding the glass in either one of these two positions, we move it up and down, and, as we move it up, note whether the horizontal arm of the cross also moves up or moves down. In either case we neutralize the apparent movement of the cross with the appropriate spherical glass (+ if the cross moves up, and — if the cross moves down). (Fig. 63.) We have thus made one meridian of our glass plane —*i. e.*, have converted the glass into a simple cylinder. Leaving the neutralizing sphere on, and still holding the glass in the same position, we now move the glass from side to side, and, as we move it to the right, note whether the vertical line of the cross moves also to the right or moves to the left. In the former case we neutralize with convex, in the latter with concave cylinders, the cylinders being added to the sphere we have already put on, and having their axes in the meridian we have already neutralized—*i. e.*, in line with the vertical arm of the cross. The sphere and the cylinder together will neutralize the glass in all meridians, and will indicate the strength of its spherical and the strength and axis of its cylindrical component.

Thus, suppose that the cross looks unbroken when we have rotated the glass until the vertical arm of the cross coincides with the meridian of 45° on the glass. The axis of the correcting cylinder must lie either at 45° or 135°. Now, moving the glass up and down—*i. e.*, strictly in line with the vertical arm of the cross—we see that the horizontal arm moves in the opposite direction, going down as we carry the glass up. A —1.00 D. spherical placed over the glass neutralizes this movement. Leaving this —1.00 D. on, we now move the glass from side to side, still keeping it with its corrected 45° meridian vertical. The vertical arm of the cross moves in the same direction that we move the glass. We now put on + cylinders with the axis at 45°—*i. e.*, in line with the vertical arm of the cross—until this movement of the cross is neutralized. Suppose it took a +1.75 D. cylinder to do this. The neutralizing glass is then —1.00 sph. $\bigcirc$ +1.75 cyl. 45°, and the glass examined is, therefore, a +1.00 sph. $\bigcirc$ —1.75 cyl. 45°.

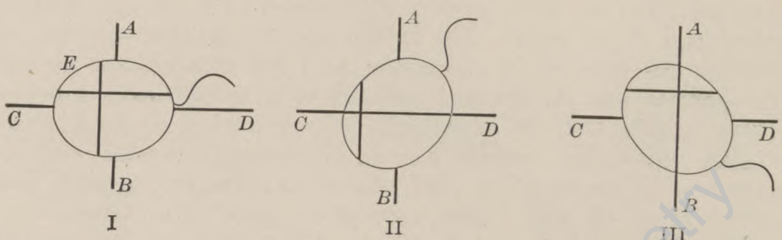
We might in the case cited have rotated the glass so that the meridian of 135° coincided with the vertical line of the cross (in which case the cross would have again appeared unbroken), and then have neutralized this meridian first. Then this correction would have worked out to —0.75 sph. $\bigcirc$ +1.75 cyl. 135°, equivalent to the one given above.

If the glass is a simple cylinder, instead of a sphero-cylinder, precisely the same procedure is adopted, only in this case we shall find that the movement along meridian is already *nil*, so that we have to apply no correcting glass to this meridian, and all that we have to do is to add neutralizing cylinders with their axes in this meridian.

If the glass contains a *prism*, one or both arms of the cross will be displaced, the horizontal arm being thrown up or down, and the vertical arm being carried to one side. As we rotate the glass, the displacement of one arm will increase and that of the other diminish. But however we rotate the glass, the arms seen through it will always remain parallel to their original direction—the vertical arm remaining vertical, the horizontal arm horizontal. Hence the cross, although appearing broken, is not distorted. That is, *a prism shifts*, but *a cylinder twists.* (Fig. 64.)

We keep on rotating the glass until the horizontal arm is no longer displaced—*i. e.*, until the portions seen outside and inside of the glass form an unbroken line. The apex of the prism contained in the glass will then lie in the direction in which the vertical line is displaced; and the point which this apex occupies on the circum-

FIG. 64.



Displacement produced by a prism. Determination of the axis of a prism. A right-angled cross, *A B C D*, is seen through a glass containing a prism (I) The apex of the prism does not lie in line with either arm of the cross, being in fact directed toward *E*. Both arms of the cross will appear displaced bodily, but will not be twisted. The amount of displacement of *AB* indicates the lateral effect, and the displacement of *C D* the vertical effect of the prism when in this position. If now the glass is rotated, both arms will appear to shift, but each will still always remain parallel to its original position. (II) The glass has been rotated until the apex of the prism points toward *C*. *C D* appears unbroken, while *A B* is displaced toward *C*, and the amount of its displacement now indicates precisely the total strength of the prism. (III) The glass is rotated 90°. The line *A B* now appears unbroken, and *C D* is deflected toward *A*. The apex of the prism points toward *A*, and the amount of deflection of *C D* corresponds precisely to the strength of the prism.

ference of the glass will be indicated by the point where the horizontal arm of the cross cuts that circumference. Thus if the apex of the prism was at 45°, both arms of the cross would appear broken when the glass was held in its ordinary position. When we rotated the glass so that its 45° meridian was horizontal, coinciding thus with the horizontal arm of the cross, that arm would appear unbroken, but the vertical arm would be displaced outward toward the 45° point on the glass's circumference.

The strength of the prism may be measured either by measuring the actual maximum displacement of one of the cross-arms on a prism-scale, such as Ziegler's (the apex of the prism being placed in line with the scale), or more readily by neutralizing the displacement with prisms placed over the glass with the apex directed the

other way. The strength of the neutralizing prism equals that of the prism in the glass.

Usually the strength of the prism that a glass contains can be determined only after neutralizing any sphero-cylinder that it may also contain.

In thus neutralizing glasses to determine the sphere, cylinder, or prism they may contain, it is essential that we look precisely through the centre of the glass examined, and also that we should hold the neutralizing glass in close contact with it.

HYGIENIC TREATMENT OF REFRACTIVE ERRORS.

In *myopia*, particularly when progressive, and especially, therefore, in children, we should be careful to give precise rules as to the amount

Fig. 65.

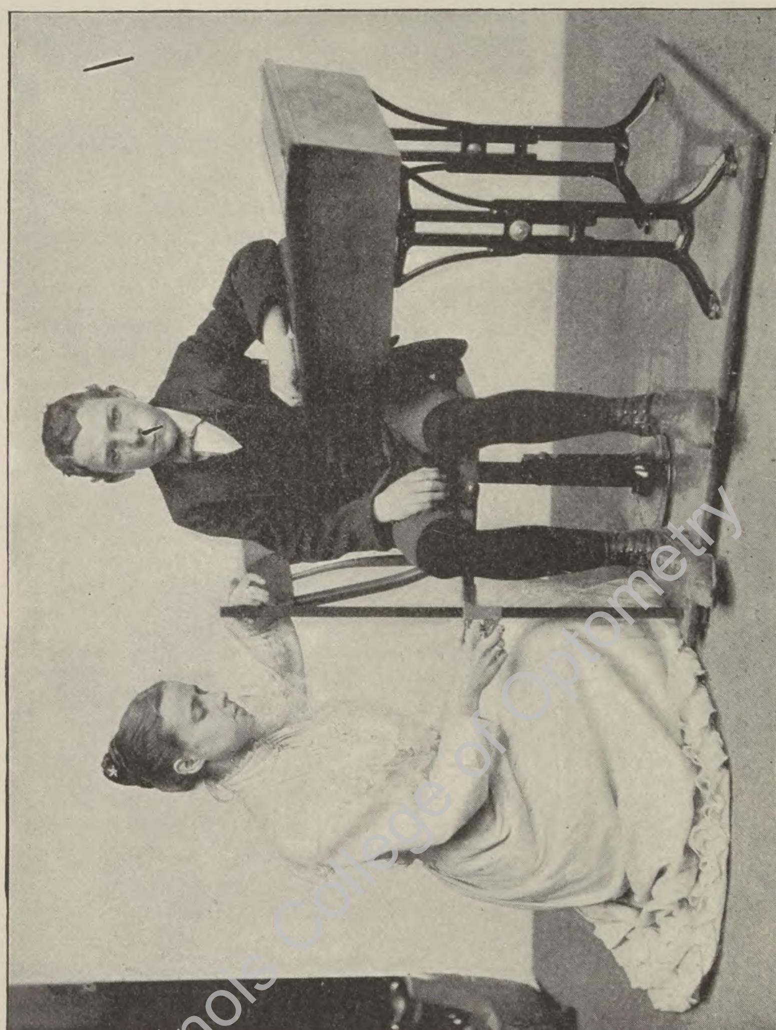
The Chandler adjustable seat and desk.

and kind of work that the patient can do, and insist upon proper lighting, proper hours of study, and plenty of sleep, with a good amount of out-of-door exercise. Use of the eyes by artificial light should be restricted, or, in the more aggravated cases, entirely forbidden. It is also a good plan to have the patient suspend his reading

or other work every fifteen minutes or so, for a few moments, and during this interval of rest either sit erect or actually stand up, and look off into the far distance.

The patient's attitude in reading or writing should be carefully looked after. He should not be allowed to bend over at his work,

Fig. 66.



Measuring the length of the leg preparatory to adjusting the height of the seat.

nor, on the other hand, should the desk be so high as to bring his work unduly close to the eyes.

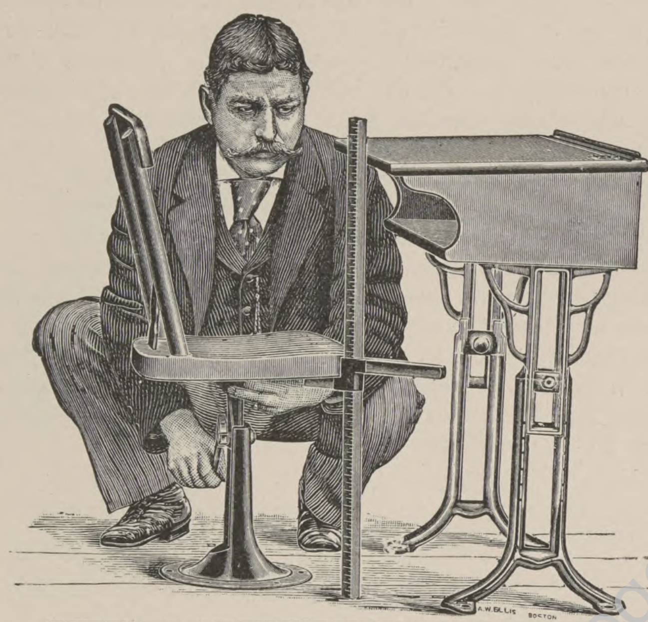
Figs. 65, 66, and 67 illustrate a form of adjustable desk well adapted for school purposes.

Those affected with progressive myopia should be discouraged from

undertaking any very fine work, such as book-keeping or sewing, requiring close and continued application. It is particularly important to have this point in mind when selecting a boy's future life-work. In very high myopia, particularly when there are symptoms of retinal irritation, total abstention from near work is often required.

In the transient myopia produced by *spasm of accommodation*, complete abstention from near work may be required, combined often with the instillation of atropine three times a day for several weeks.

FIG. 67.



Adjustment of seat.

In all conditions of refraction the *illumination* should be attended to carefully. The ideal illumination is that afforded by diffuse daylight. When artificial light is used, this also should be as diffuse as possible. Hence, it is a mistake to have a brilliant light, particularly a shaded light, over the desk, and have the rest of the room in comparative darkness. There should be at least one other light in the room, and more, if necessary, so that the illumination may be pretty evenly distributed all over it. The brilliancy of an electric or a Welsbach light often needs tempering, and this is best done by a very light amber or yellowish shade.

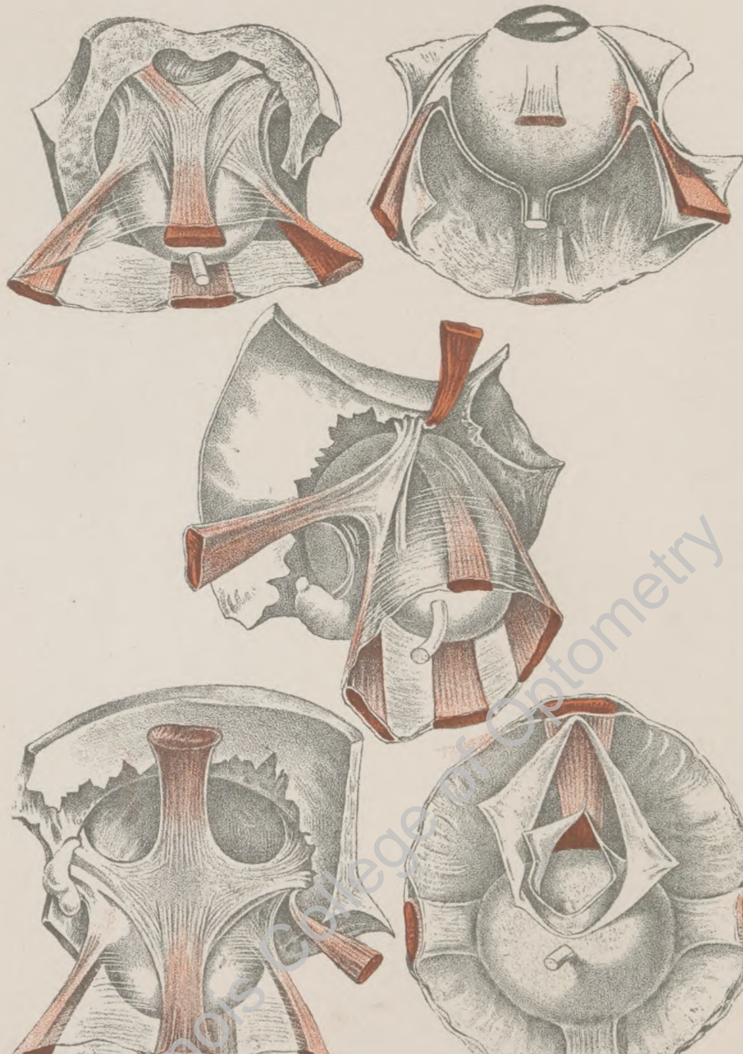
Patients complaining of *photophobia* should not be permitted to wear smoked or colored glasses unless there is actual disease of the fundus, or unless the photophobia is due to some quite temporary

cause, as dilatation of the pupil from instillation of atropine. Otherwise, the abnormal sensitiveness to light will be aggravated by the use of the glasses, and the patient become more and more incapable of using the eyes.

In *hyperopia* and *astigmatism* we sometimes, on account of the asthenopia and other symptoms, have to diminish the amount by which the eyes are used, and increase the amount of open-air exercise. But, in general, hyperopia and astigmatism furnish no absolute bar to eyework, and it is usually a distinct mistake to yield too much to the patient's complaints. We should not, in the absence of organic disease of the fundus, encourage the patient to *disuse his eyes* simply because the use of them is painful. By so doing we may initiate a vicious circle, and progressively increase the patient's disability, instead of removing it. On the contrary, moderate, systematic, and increasing use of the eyes for near work is the rule in such cases. The ophthalmologist himself should give careful and precise directions as to the amount and kind of eye-work to be done, and determine by experiment how rapidly the work may be increased.

Finally, it should always be borne in mind that in treating refractive errors we must constantly have regard to the *general condition* of the patient and to the state of the organs other than the eyes. In many cases in which the symptoms seem to be due entirely to the eyes, treatment directed to the general condition, to the nose and throat, or to the pelvic organs, will often relieve an asthenopia which glasses have failed to relieve, and in not a few instances will render it unnecessary to use glasses at all.

PLATE III.



Capsule of Tenon. (Motais.)

CHAPTER IV.

THE MOTIONS OF THE EYEBALL AND THEIR DERANGEMENTS.

By CASEY A. WOOD, M.D.

Anatomy and Physiology. The ocular muscles are divided usually into *internal* or *intrinsic*, and *external* or *extrinsic*. Although it is necessary, for convenience of reference, to speak of the individual extrinsic muscles, both the single and associated excursions of the eyes may be regarded, in every case, as compounded of movements due to actions of all of them. The *iridic* and *ciliary* muscles comprise the first class, while six others, the external rectus, internal rectus, superior rectus, inferior rectus, superior oblique, and inferior oblique, make up the second. These, with the single exception of the inferior oblique, form a cone, whose apex points toward the foramen opticum, while its base envelops the eyeball in front of the equator. According to Weiss, if the axes of the orbits are projected backward, they form an angle of from 20° to 37°, depending upon race, age, and the peculiarities of the individual. These considerations also govern the conformation of the muscular cone, affect the degree of its divergence, and may even determine the shape of the eyeball. (Plate III.)

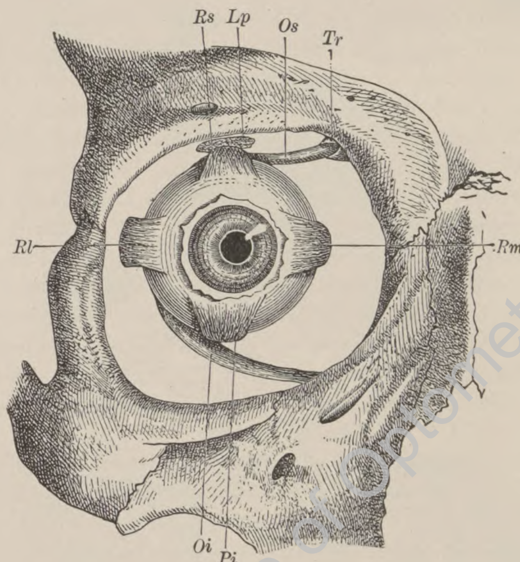
The globe itself has a *centre of rotation* around a point in its antero-posterior axis. In the emmetropic eye this is about 14 mm. behind the cornea and 10 mm. in front of the posterior surface of the sclera. The *primary position of the eye* is that in which, when the head is held erect, the gaze is directed straight forward in the horizontal plane. The vertical meridians of both eyes are then exactly vertical and parallel. It is from this starting point that the movements of the eyeball are considered.

The innervation of the extrinsic muscles of the eye is easily remembered. All of these, as well as the levator palpebræ superioris, the ciliary muscle, and at least one of the iris muscles, are supplied by the third nerve, except the external rectus, which is supplied by the sixth nerve, and the superior oblique, which is supplied by the fourth.

While we recollect that every excursion of the globe is attended by more or less contraction or lengthening of most of the extrinsic muscles, and sometimes by action of the internal muscles, we have to speak of the probable action of the individual extrinsic muscles. The observations of Swanzy appear to give the most satisfactory explanation of the various and complicated movements of the eyeball,

both separate and associate, and consequently they are adopted for the purposes of this chapter. Donders proved experimentally that it is only in the primary position and when the eyes are turned directly outward or inward, downward or upward, that the vertical meridian maintains its vertical direction. When the eyes are turned in other directions, there is always a sort of wheel-motion given to the globe, and the vertical meridian of each normally balanced eye is inclined at about the same angle. These various positions of the vertical meridian can be understood best by a reference to the actions of the various muscles in the associated movements of the eye and by a reference to the figures. The author just quoted points out:

FIG. 68.



Showing attachment of the orbital muscles. *Rl.* External rectus. *Rs.* Superior rectus. *Lp.* Levator palpebræ. *Os. Tr.* Superior oblique with its pulley. *Rm.* Internal rectus. *Oi.* Inferior oblique. *Pi.* Inferior rectus.

1. In the primary position all the muscles are practically at rest.

2. Motion of the eyeball directly outward is effected by the external rectus alone, and motion directly inward by the internal rectus alone.

3. Motion of the eyeball directly upward and directly downward is effected mainly by aid of the superior and inferior recti. At the same time these muscles, acting alone, also rotate the eyeball directly inward and give a certain inward inclination to the vertical meridian, which, in this position, should be upright. Consequently, in rotation of the globe directly upward the inferior oblique, which rotates the eye slightly outward as well as upward and inclines the vertical meridian outward, must be associated with the superior rectus in order to

counteract, in these particulars, the tendency of its action. In rotation of the eyeball directly downward, the inferior rectus must be associated with the superior oblique, which acts antagonistically to this straight muscle, in respect to rotation inward and to outward wheel-motion.

4. Rotation upward and outward is effected chiefly by aid of the superior rectus and external rectus; but the latter muscle has no influence over wheel-motion, while the former produces wheel-motion inward; yet the inclination of the vertical meridian is outward in this position, and therefore a third muscle, which will supply this inclination in a high degree, is required, namely, the inferior oblique, whose power over the wheel-motion of the eyeball is greatest when the latter is turned upward and outward.

5. Rotation downward and outward is effected chiefly by the rectus inferior and the rectus externus. Inasmuch, however, as the former inclines the vertical meridian outward, while the latter has no influence over it at all, a third force is required which will bring about the necessary inward wheel-motion, namely, the superior oblique, whose influence in this respect is most powerful when the eye is turned downward and outward.

6. Rotation upward and inward is brought about chiefly by the rectus superior and the rectus internus; but the effect of the former upon the inward wheel-motion of the eye would be so great as to interfere with parallelism of the vertical meridians of the two eyes, that of the other eye not being inclined outward in a corresponding degree. A third force, therefore, is required which will, to a certain extent, counteract the influence of the superior rectus in this respect, and this is found in the inferior oblique, which, in this position of the eyeball, has but slight power over its wheel-motion.

7. Rotation downward and inward is chiefly the result of contraction of the inferior rectus and the internal rectus. The power of the former over the outward inclination of the vertical meridian would, in a similar way, be too great, and must be similarly corrected by the action of the superior oblique.

The position of rest is probably divergence and, in all probability, even a slight degree of convergence, as well as parallelism of the ocular axes, is maintained by more or less effort.

The internal rectus arises from the tendon common to it and the inferior rectus, at the inner aspect of the optic foramen, and runs forward close to the inner wall of the orbit, to be inserted into the sclera by a tendinous expansion 9 mm. long and 10½ mm. wide, 6½ mm. from the sclerocorneal junction.

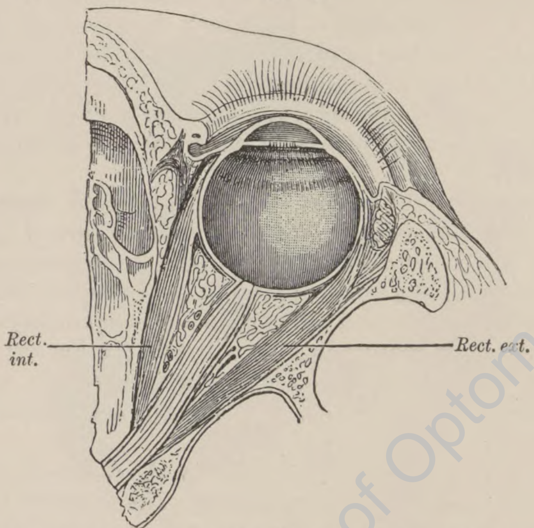
The external rectus arises from the greater wing of the sphenoid by two heads that become united and form the body of the muscle, which runs forward close to the external wall of the orbit. It is attached to the sclera by a tendon, 3.8 mm. long and 9.5 mm. wide, about 7 mm. from the limbus corneæ.

The inferior rectus arises from the tendon common to it and the internal rectus, and runs forward on the floor of the orbit, and is attached to the globe by an aponeurosis 6 mm. long, 9 mm. wide, and about $7\frac{1}{2}$ mm. from the sclerocorneal junction.

The superior rectus arises from the upper edge of the optic foramen from the common tendon. It passes forward just beneath the levator palpebræ superioris and is inserted into the sclerotic 8 mm. from the sclerocorneal junction. Its tendon is 6 mm. long and $10\frac{1}{2}$ mm. wide.

The superior oblique arises from the lesser wing of the sphenoid, passes forward along the inner wall of the orbit, where it becomes tendinous and forms a pulley-like adjustment which plays within a fibrous ring situated in the trochlear fossa. The direction of the

FIG. 69.



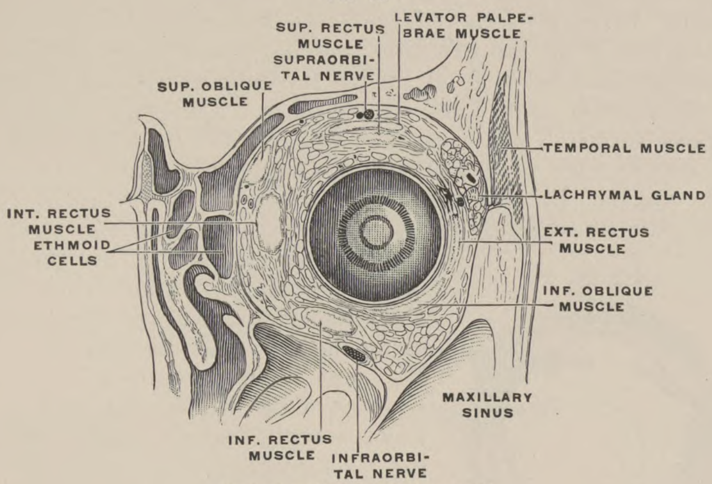
Horizontal section of the eye in the orbit, showing the relative attachment of external and internal recti.

muscle now changes, and it proceeds backward, downward, and outward at an angle of 55°, passes beneath the superior rectus, and is inserted into the sclerotic between the cornea and the optic nerve about 17 mm. from the corneal limbus. The width of the aponeurotic insertion of this curious muscle into the globe varies from 7 mm. to 15 mm. It is one of the principal factors in globar rotation, turning the upper part of the vertical meridian inward.

The inferior oblique arises from the anterior third of the floor of the orbit, in a pit situated in the superior maxillary bone. It then proceeds outward, backward, and upward, beneath the inferior rectus muscle, to be inserted into the sclerotic between the inferior and external recti on the posterior half of the globe, $17\frac{1}{2}$ mm. from the sclerocorneal junction.

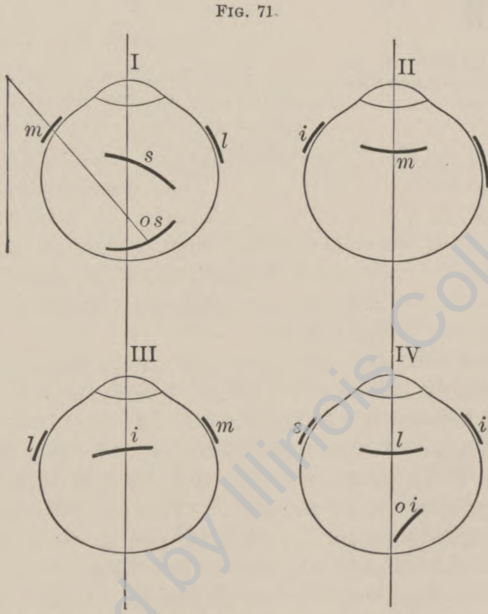
Tenon's Capsule. That we may the better understand the attachments of the various extrinsic muscles, it is necessary to say something about the closed sac known as Tenon's capsule. This mem-

FIG. 70.



Vertical section through the orbit.

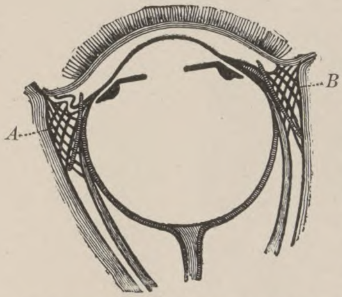
FIG. 71.



Schematic representation of the insertion of the extra-ocular muscles into the globe, showing the distance of the same from the corneal limbus. I. Superior aspect of the globe. II. Medial aspect. III. Inferior aspect. IV. Lateral aspect. *s.* Rect. sup. *i.* Rect. inf. *m.* Rect. int. *l.* Rect. ext. *os.* Obl. sup. *oi.* Obl. inf.

brane envelops the greater portion of the eyeball, and is united behind, as well as in front, to form a closed cavity. It is attached close to the optic foramen, and, passing forward, becomes loosely adherent to the sclerotic. Anteriorly it is attached to the conjunctiva by connective tissue, and extends to within 3 mm. of the corneal junction. Its cavity forms a large lymph space lined by endothelium. The tendons of all the extrinsic muscles, the obliques behind and the straight muscles in front, pierce the capsule without interfering with its function as a closed lymph space. The ciliary nerves likewise pass through Tenon's capsule, which, let it be noted, also communicates through the sclera with the suprachoroidal and perineural lymph spaces. In this way it connects the intercorneal lymph spaces with the exterior of the eye. When a muscular tendon or nerve trunk passes through this membrane a portion of the latter extends along its external surface, thus strengthening their connections with the sclera. We may then regard the combined tendons of the straight muscles, in conjunction with Tenon's capsule, as forming a complete envelope about the eyeball, and this fact must be reckoned with in operating on the former. An incision sometimes fails to reach the muscular tendon, because both layers of the capsule have not been divided, and because, also, it is not borne in mind that prolongation from the capsule itself and what are known as the "check ligaments" may interfere materially with the results desired. (Fig. 72.) This is especially the case when these ligaments are abnormally developed. It is not an uncommon experience to find, even after the most complete division of the tendon, that relative positions of the globes are unaffected because of these connections between the muscular tendons, the capsule of Tenon, and the check ligaments.

FIG. 72.



A. Internal check ligament. *B.* External check ligament. (HANSELL and REBER.)

As a rule, however, the action of the check ligaments is a normal one; they probably prevent or retard overaction of the adductors and abductors, as shown in Figs. 73 and 74.

The blood supply of the muscles is derived from the ophthalmic artery, through the minute muscular branches, shortly after it enters the orbit through the optic foramen. These capillaries are tortuous and loosely attached to their surroundings, so that the movements of the globe and the shortening and lengthening of the muscles themselves do not interfere with their continuity. It sometimes happens that cutting of these small vessels gives rise to profuse hemorrhage, but this is rarely, or never, a serious matter. The arteries are accompanied by veins of the same name.

Nerve Supply. It is desirable to say something further about the innervations of the various muscles. The areas in the cortex associated with the movements of the eyeball are not, as yet, precisely located. They are probably in front of the large motor area, close to the neuron that presides over the facial muscles, and undoubtedly have a near association with Broca's speech centre. The basal nuclei are regarded generally as lying well within the gray matter in the aqueduct of Sylvius and on the floor of the fourth ventricle, most of them just beneath the corpora quadrigemina. According to the arrangement of Stuelp, the subcortical nuclei of the internal eye muscles are situated in the anterior portion of the third nerve nucleus. These fibres supply the ciliary muscle and the sphincter iridis. Immediately hehind them are the nuclei of all the external muscles supplied by the third nerve. Still further back are the nerve cells which constitute the nucleus of the fourth nerve that supplies the superior

FIG. 73.

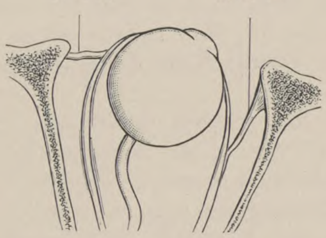


FIG. 74.

FIG. 73.—The check ligaments during partial contraction of the external rectus muscle, the internal check ligament (*I. C. L.*) being in a state of maximum relaxation, and the external (*E. C. L.*) somewhat stretched. (MOTAIS.)

FIG. 74.—Diagram intended to show how, during full contraction of the external rectus, the external check ligament (*E. C. L.*) is stretched to its maximum length, and the internal (*I. C. L.*) is slightly stretched also. (MOTAIS.)

oblique; and, finally, still behind these are the nucleoli of the sixth nerve that governs the external rectus. All of these nerve centres are connected with one another by nerve fibres that run from one nucleus to the other. It should not be forgotten that minute nervous connections probably exist between the motor neurons governing the various eye muscles, and all the other nerve centres.

That the individual may fix both eyes with ease upon objects distant and near, and *obtain binocular vision in all parts of the field of vision,* it is necessary that a certain balance exist between the forces that rotate the eyes in various directions. Moreover, both eyeballs must be *steadied* in their fixation. We must believe consequently that all the external muscles are, during the waking hours, in a state of unconscious tonic contraction, and that every act of sight is accompanied by more or less expenditure of nerve force.

Convergence. A state of divergence of the visual axes being the probable condition of rest, parallelism and convergence require more

or less nervous effort. The axes may, however, be made to cross until they form a very wide angle, estimated to vary in individuals from 45° to 65°. When the eyes converge there is not only contraction of the internal rectus, but of the superior and inferior recti, and of the iridic and ciliary muscles. It is very likely, also, that other muscles of the eye, as before noticed, take part in this act of convergence by a sort of modified contraction, so as to steady the eye. One method of measuring convergence is by means of a prism, apex in, placed before either eye—prism convergence. (See page 159.)

Divergence. It is very likely that in a state of absolute rest the visual axes diverge 8° to 10° from parallelism, and this may be demonstrated by making use of prisms, apex out. Indeed, it may with truth be affirmed that convergence should be regarded as beginning at this point. Hence, we have the term *negative convergence*, because from a state of divergent rest we proceed to parallelism and then to convergence. The nearest point at which a very small object can be seen singly, with both eyes, constitutes the *fusion near point*. The divergent power in any individual may be measured by finding the highest prism, apex out, that can be held before either eye without producing double images of a candle flame 6 m. distant—prism divergence.

Deorsumvergence. When the eyes are turned downward it is mainly, as we know, through the contraction of the superior oblique and the inferior rectus, and this act is termed deorsumvergence.

Sursumvergence refers to the act of turning the eyes upward.

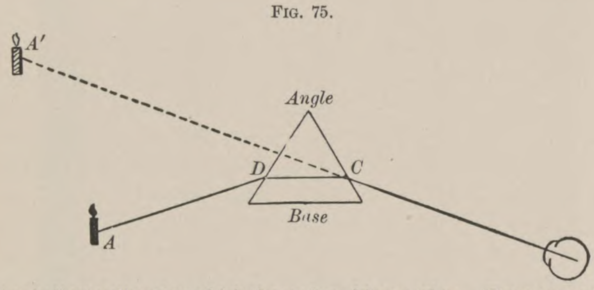
Associated Movements. So far, reference has only been made to the fact that it is necessary for binocular vision that both eyes be rotated in precisely the same direction. With properly balanced muscles, every movement of one eye is associated with an instant and equal rotation of the other eye. Thus, it is impossible to turn one eye up and the other down, or both eyes outward, as that would produce an annoying double vision and interfere with binocular sight. On the other hand, owing mainly to an arrangement of the nerve fibres in the central neurons presiding over the various eye muscles, the associated movements of the eyes are so arranged as instantly to fix corresponding retinal points upon images perceived by the brain.

Accommodation-convergence. With every effort of convergence there goes a certain amount of accommodation. Although within reasonable limits accommodation and convergence are constant, one may be increased or diminished a little with reference to the other without disturbing symptoms. For example, before the normal eye of an individual a concave glass may be placed, and yet, with a slight effort, an object at, say, three feet distance will be readily and distinctly seen. In the same way a convex glass still enables a person before whose eye it is placed to see, with both eyes together, the same object distinctly and at the same distance. For the more definite measurement of convergence efforts the term *metre angle* is

used. This is the angle which the visual line makes with the median line of the face, the latter being drawn at right angles to the base line (interocular base line) joining the centres of rotation. The angle formed by the median line and the visual line at a point 1 m. distant from the base line is termed 1 metre angle. An object seen $\frac{1}{2}$ metre distant from the eye would require twice as much convergence as that seen at 1 m.; convergence, therefore, at this point, would be 2 metre angles; at $\frac{1}{3}$ m. distance it would be 3 metre angles, at $\frac{1}{4}$ m., or 25 cm., it would be 4 metre angles, etc. On the other hand, an object seen at 2 m. distance would require only one-half the amount of convergence to fix it at 1 m.; convergence here, then, equals $\frac{1}{2}$ metre angle—in other words, C. = $\frac{1}{2}$ m. a. *Where both eyes are normal and emmetropic, as many metre angles of convergence are required as there are dioptres of accommodation.*

The *angle gamma* is that formed at the centre of rotation by the optic axis and the line of fixation. It should not be confused, as it sometimes is, with the *angle alpha* formed at the nodal point by

FIG. 75.



Dotted line indicates direction which the projected beam takes. (HANSELL and REBER.)

the visual axis and the major axis of the corneal ellipse. In measuring the angle gamma, it is well to make use of the perimeter. The patient is placed in the primary position, with his chin on the chin-rest and gazing at the fixation point. A small candle is moved along the perimeter arm until it is reflected from the exact centre of the cornea into the eye of the observer gazing at it immediately behind the flame. This point is read off in degrees upon the perimeter arc. The angle varies from 4° to 6°.

Prisms. One cannot properly understand the contractile or rotary power of the eye muscles without having a proper idea of the effect of prisms upon beams of light and upon the position of the images cast upon the retina. A prism is a piece of glass whose refracting surfaces are inclined toward each other so as to form an angle whose degree is commonly employed to designate its particular prism. This also forms its edge or apex. The angle subtends the thicker portion or base of the prism. A ray of light, instead of passing through unchanged in direction, is bent in its course from the perpendicular toward the base of the prism, and if the eye be placed in the path

of the light ray after it has passed through the prism, the luminous body from which it emanates will not be seen in its true position, but in the direction of the visual line projected beyond the glass. This is in accordance with the law of projection. The prism projects the ray of light to a false position on the retina, and the retinal elements perceive the object as coming from a straight line, the continuation of the visual axis. When a prism is placed in front of one eye, and the visual image is thus projected upon an unusual part of the retina, *an effort is at once made by the nerve centres so to adjust the eye that binocular vision may result.* If the apex of the prism be directed immediately inward, the task of accomplishing this is laid upon the muscles that rotate the eye inward—the adductors, the chief of which is the internal rectus.

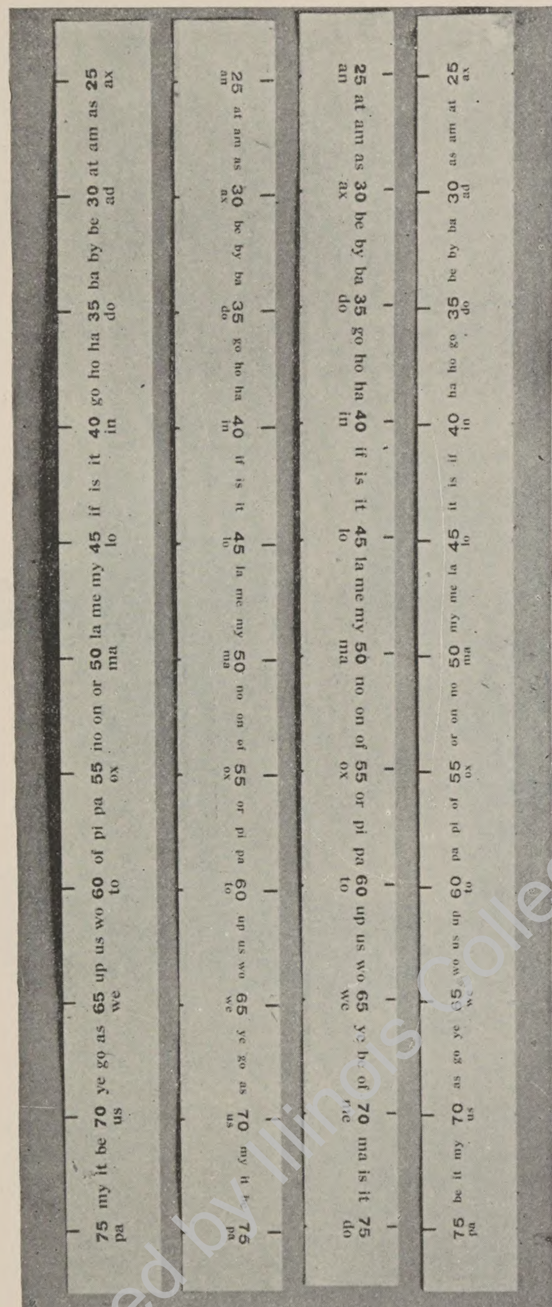
We know from experience that after a few trials the adductors of normal eyes can overcome in this way the diplopia that would otherwise be produced by prisms as high as 40° or 50°. Normal *adduction* is thus said to be about 40° or 50°. On the contrary, when the apex of the prism is directed immediately outward before one of the eyes, the task of overcoming the diplopia must be made by the muscles that rotate the eye outward, chiefly the external rectus. *Abduction* measured by prisms is in the normal eye about one-fourth that of adduction, namely, 10°. In the same way we find that *infraduction* and *supraduction* (prisms up or down before each eye) may be measured in terms of the power required to overcome the diplopia that would otherwise be produced. This varies from 2° to 4°.

Abduction, as found at the first office examination, is fairly constant, and in normal eyes will rarely fall below 7°. The ratio between adduction and abduction (prism divergence) for twenty feet ranges from 8:1 to $2\frac{1}{2}$:1. No arbitrary standard can be fixed as yet, simply because the figures thus far offered have been largely a matter of personal equation.

Field of Fixation. An examination of the movements of the eyeballs should determine the rotating powers of the muscles of each eye. When these extreme points have been found and connected, they constitute the *monocular field of fixation.* The writer,[1] several years ago, adjusted the perimeter to mark out this territory. Instead of having an object attached to the carrier on the perimeter arm in the usual way, he used a simple device whereby the rotation of the globe in any direction is rapidly and easily measured. Four strips of unglazed parchment paper have printed on them words of two letters corresponding to Jaeger XI. at 50 cm., placed between as well as below figures representing the degrees of latitude on the perimeter arc. These strips are together placed in position on the arm of the perimeter (Fig. 76), the patient's head being in the primary position, and he is asked to read the lower line as far away from the centre as possible. This accomplished, he is requested to

[1] Journal of the American Medical Association, 1896.

FIG. 76.

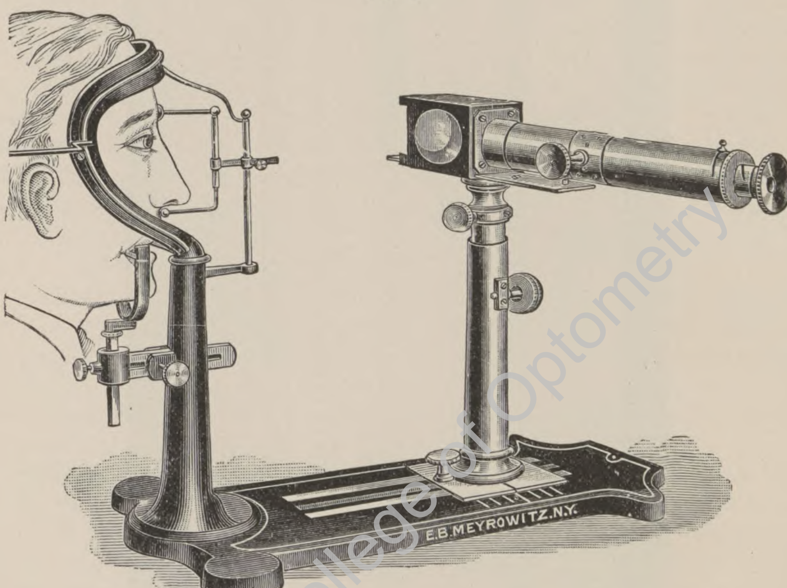


Scheme for the measurement of the monocular field of fixation by means of the perimeter. (CASEY WOOD.)

give the figure placed above the word just recognized, and to try to read farther out additional letters on the figure line. Each word beyond represents about one degree on the scale, and the number of words so read added to the previous figure gives the limit, in degrees, of the field in that direction. As each quadrant of the circle is passed over, a slip of paper is removed, revealing the next paper, whose lettering, being different, suggests nothing to the person under examination. After many perimetric examinations with this device, the limits of the monocular fixation field were found to correspond closely with the figures of Landolt:

Directly out	45°	90°	Out and down	47°	92°
Directly in	45°		In and up	45°	
Directly down	50°	93°	Down and in	38°	85°
Directly up	43°		Up and out	47°	

FIG. 77.



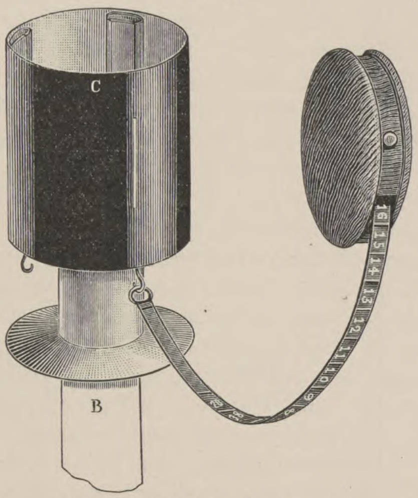
Stevens' tropometer.

The binocular field of fixation is by no means identical with the field of binocular single vision, although its borders are about the same. Duane finds that the upward limit of the monocular field to be about 45°, downward about 70°, right and left 55°. These rotations vary with age and with the form and position of the globe. Doubtless the best method of measuring the rotations of the eye, and, consequently, the field of fixation, is by means of Stevens' tropometer. (Fig. 77.) As we shall hereafter find, it is necessary to know, so far as it can be determined, whether a particular

muscular imbalance is due to weakness or spasm of some particular muscle or muscles. For this purpose, the rotation power of the globe in all directions as measured by the methods just described will be found of extreme value.

Another useful instrument for the *measurement of convergence and its relation to divergence* is the Landolt ophthalmodynamometer. In it we possess a method comparable to the measurement of the amplitude and range of accommodation by noting the relative position of the *punctum proximum* and the *punctum remotum* of convergence. This device consists of a black metal case fitted over a candle. This cylindrical case is pierced by various openings, the most valuable of which is a narrow slit through which the candle-light can be seen as a streak of light. Just below each one of the openings is a hook to

Fig. 78.



Landolt's ophthalmodynamometer.

which is attached a tape-measure marked in centimetres, and on the other side the metre angles corresponding to them. The candle is held directly in front of the patient's face, gradually approaching it, until the light streak becomes doubled. The tape indicates in centimetres this distance (which is the punctum proximum of convergence) as well as the amount of convergence in metre angles. We have seen that the number of centimetres distant from the interocular base line to the point of doubling divided into 100 gives the number of metre angles of convergence. If the double vision occurs at 10 cm., we know that the patient possesses 10 metre angles of convergence. The punctum remotum of convergence may be obtained by having the patient, with his head in the primary position, fix a point of light, say, a candle flame, at 6 m. and find the strongest prism, apex out, before one eye, that can be overcome

without producing diplopia. We then divide the number of that prism in degrees by seven, and thus obtain in metre angles the amount of *negative* convergence of each eye. If the prism be 6°, he has 0.85 metre angle; if 7°, 1 metre angle, and if 9°, 1.28 metre angles, and so on. The average amount of convergence in the normal condition is about 10 metre angles.

Many are the devices that, in addition to those mentioned, have been employed for estimating the excursion powers of the ocular muscles. Probably the best of these are the Risley rotating prism, the Maddox rods, the monocular phorometer of Savage, and the Gould and Noyes prism batteries.

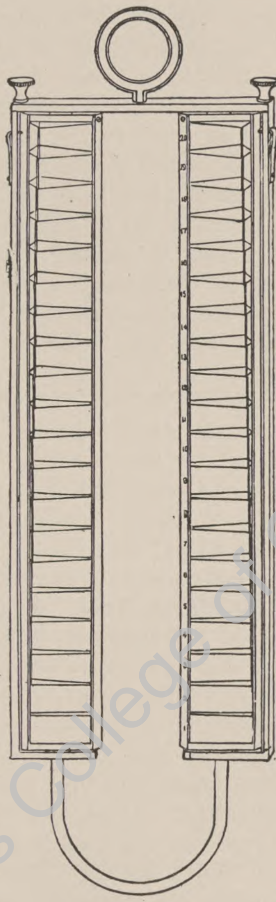
Heterophoria, Heterotropia. Deviations from the normal balance of the eye muscles have received various names, and we have only to mention *insufficiency* or *weakness of convergence* to indicate one phase of what Gould has very properly termed "muscular imbalance." The nomenclature of Stevens has been most widely accepted. Normal muscle balance he terms *orthophoria;* abnormal balance, or imbalance, heterophoria. *Hyperphoria* is a tendency of the visual axis of one eye to deviate above that of the other; *hypophoria* is a tendency of the visual axis of one eye to deviate below that of the other; *exophoria*, a tendency of the visual axes outward; *esophoria*, a tendency of the visual axes inward; *hyperexophoria*, a tendency of the visual axis of one eye upward and outward; *hypoexophoria*, a tendency of the visual axis of one eye to deviate downward and outward; *hyperesophoria*, a tendency of the visual axis of one eye to deviate outward and inward; *hypoesophoria*, a tendency of the visual axis of one eye to deviate downward and inward. To this list, Savage has added insufficiency of the oblique eye muscles, or *cyclophoria;* and Duane, a defect of power of some particular eye muscle—*hypokinesis*—excess in action of a particular eye—*hyperkinesis*—while irregular action of an individual muscle is styled *parakinesis*. Where the visual axis exhibits something more than a *tendency* toward abnormal excursion, the termination "tropia" is used, instead of "phoria;" thus, in *exotropia* the visual axis, as compared with that of the opposite eye, does visibly turn outward. It sometimes happens that in the same individual there may be, for example, a hyperphoria for near fixation, and a manifest hypertropia when he gazes in the distance.

The term "insufficiency of the ocular muscles" corresponds to von Graefe's *dynamic strabismus*. The test which in von Graefe's time was commonly made of the convergence power is a simple one: The patient is asked to fix an object held directly in front of his face, at a distance of 1 m. This is gradually approached to within 10 or 15 cm. of the eyes. If there be weakness of convergence, the eye with the weaker internal rectus usually turns out. Another well-known and useful test is to ask the patient to fix a dot upon the card held a little below the horizon, 25 cm. from the eyes. One eye is then covered, and the other

eye is watched to determine whether it, behind the cover, deviates outward, inward, upward, or downward. If there be no deviation after first one eye, and then the other, has been covered and uncovered, one may decide that there is little or no imbalance of the muscles.

For testing the functions of the muscles that produce vertical excursions, a 10° or 15° prism is placed, base in, before either eye, taking care that the prism axis is exactly horizontal. If the double images thus produced at 20 feet or 6 m. are on the same level, there is no hyperphoria. In much the same way, *the functions of the lateral muscles* may, under similar conditions, be tested. A 7° or 8° prism is placed base up or down, before one eye. This produces vertical diplopia. If one image is seen directly over the other, there is no excess of divergence or convergence.

FIG. 79.



Gould's prism battery.

There are several methods of testing the *balance of the muscles at the near point* or ordinary working distance, which varies according to the occupation from 25 cm. to 40 cm. For all practical purposes we may employ it at 30 cm. A card having a small dot and a fine line drawn through the latter is placed at this distance, just below the horizon. Vertical diplopia having been produced in the manner just mentioned, the double images will be found, in equilibrium, to stand directly above the other. When crossed or homonymous deviation is produced, we know that there is insufficiency or excess of convergent power, and the prism that restores the images to their normal position represents the amount of imbalance.

Another useful method of determining the *power of adduction* or prism-convergence, abduction or prism-divergence and sursumduction, is by finding the strongest prism which the lateral and vertical muscles can overcome.

Adduction is tested when the patient, with his head in the primary position 6 m. from a candle or other flame, endeavors to overcome the double images produced by a prism, apex in, placed before one or the other eye. One should begin with a weak prism, and gradually increase it until the diplopia is such that no effort on the part of the patient succeeds in fusing the double images. It should be remembered

that the adductive power is often greater by several degrees when the prism is placed before one eye than when it is held before the other; that the images should always be kept in the same horizontal line; and that the eye should be encouraged to fuse them by bringing the candle flame to within 2 or 3 feet of the patient's face and gradually carrying it to infinity, or 20 feet distant. Another method of inducing fusion, and thus measuring the total adducting power, is to ask the patient to fix the end of his finger, held 20 cm. in front of his eyes, and then, immediately afterward, to gaze at the more distant candle flame. The average of adducting power at the first trial varies from 30° to 50°, in patients with normal eyes. Gould and Lippincott have described the space between the least and greatest power of adduction as the "region of diplopia."

The power of abduction is in the same way tested with a prism horizontally placed; it will then be found that in normal eyes a prism of from 6° to 8° will be overcome. In measuring sursumduction, a prism is placed with its base up before one eye; the highest degree that can be overcome is noted. In all examinations made at 6 m., the ametropia should be corrected for the distance, and, in the same way, when a near test is employed, reading-glasses should be used.

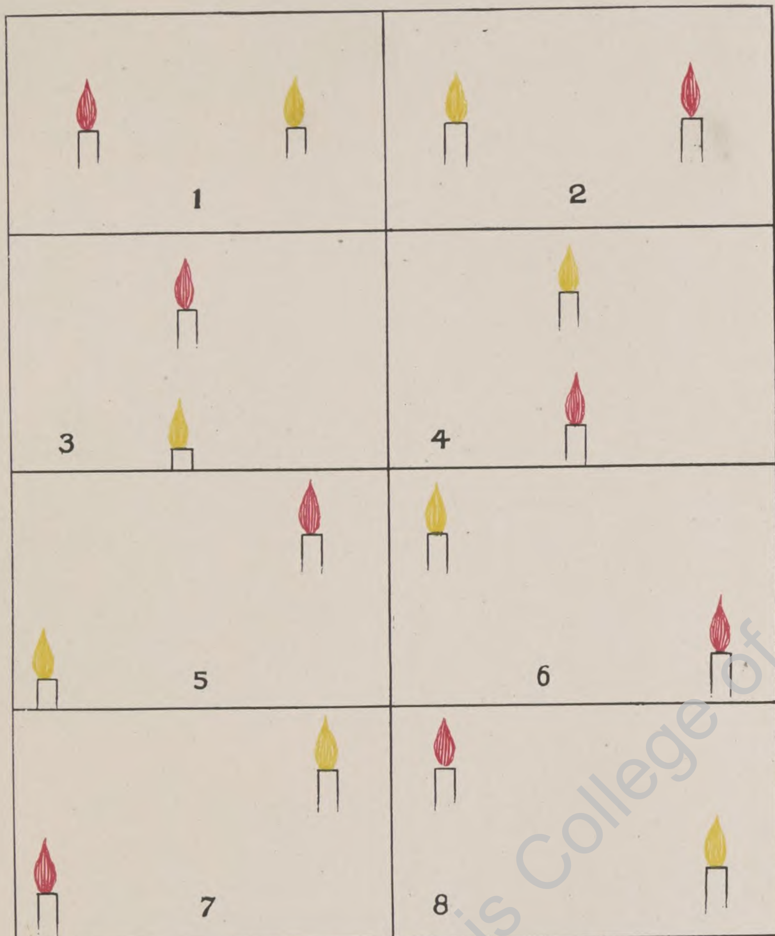
A very convenient and useful method of gradually increasing the strength of the prisms in measuring the power of rotation possessed by the extrinsic muscles in a particular case, is the *prism battery* of Gould, modified from the one first suggested by Noyes. (Fig. 79.) A series of prisms increases on each side of a central slide by 1° up to 20°, then by 2° up to 40°. The prisms may be revolved upon a pivot so as to present bases up, down, in, or out.

Red Glass Test. If a red glass be placed before one eye, binocular vision is overcome in most cases of heterophoria, and two images, one red and the other yellowish, of a candle or other flame are observed. The relative positions of these lights is a fairly reliable indication of the form and amount of the imbalance; indeed, it furnishes an easy though rough test of the heterophoria present. The prism needed to fuse the two images furnishes the degree of the latent deflection. (Plate IV.)

Instruments used in testing the balance of the extrinsic eye muscles are based mainly upon the foregoing methods. That is to say, the impulse for binocular fixation is nullified as far as possible, so that each eye may be rotated to a point which represents the strength of its muscles with other factors eliminated.

The Phorometer of Stevens. Double vision is produced by a prism, base down or up, before one eye, for the examination of the power of the lateral muscles, another prism, base in or out, being used for measuring the power of the vertical muscles. It is, in effect, a more accurate application of the prism tests just described, and may be employed for examination both for the distant or the near point.

PLATE IV.



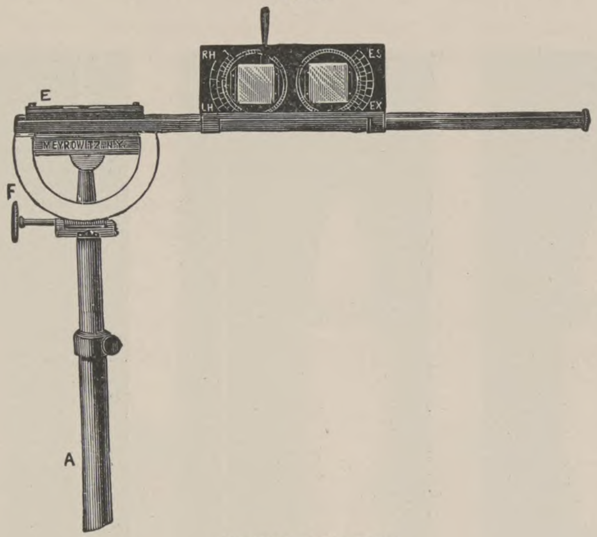
Red Glass over Right Eye.

1. Exophoria.
2. Esophoria.
3. Left Hyperphoria.
4. Right Hyperphoria.
5. Left Hyper-esophoria.
6. Right hyper-esophoria.
7. Right hyper-exophoria.
8. Left hyper-exophoria.

(Colburn.)

Knowing its limitations, and allowing for its liability to error, it is probably the best instrument that we possess for measuring heterophoria.

FIG. 80.



Stevens' phorometer.

The Risley Prism, consisting of two superimposed prisms, with their bases in opposite directions, may be used with the trial frame. This is an application of Herschel's plan, who showed that by rotating two prisms in opposite directions we can produce the effect of a single increasing prism. With this device the amount of adduction, abduction, and sursumduction may readily be measured.

FIG. 81.

Risley's rotary prism.

FIG. 82.

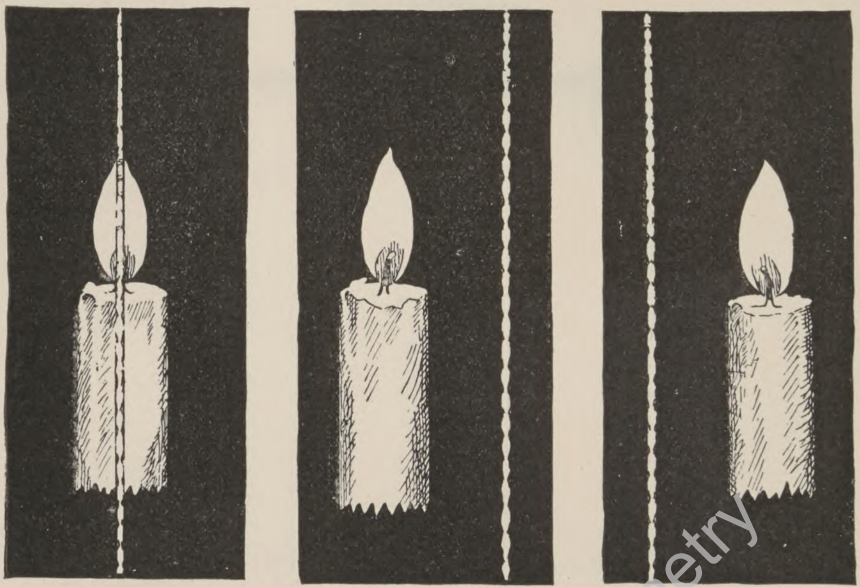
Maddox multiple rod.

The Maddox simple or compound rod is preferred by many, because the line or streak of light that appears to the eye looking through it, is less likely to be fused than are two similar light-objects when diplopia is brought about in the usual way. In orthophoria,

the candle flame (seen as a streak of light by the eye before which the rod is placed) passes through the candle flame seen by the other

FIG. 83.

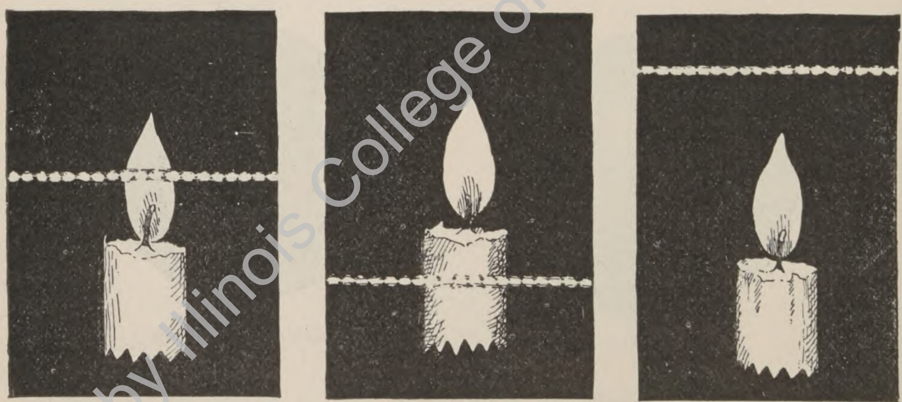
A B C



Maddox's rod-test for horizontal deviation; the rod is before the right eye. *A*. The line passes through the flame—orthophoria. *B*. The line passes to the right of the flame latent—convergence or esophoria. *C*. The line passes to the left of the flame—latent divergence or exophoria. (DE SCHWEINITZ and RANDALL.)

FIG. 84.

A B C

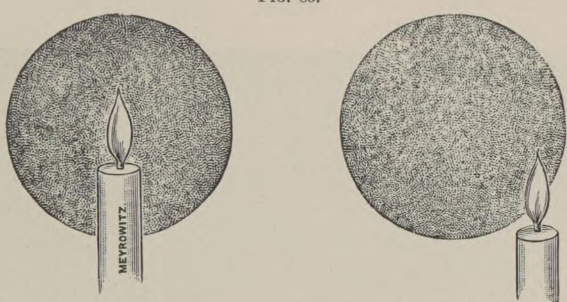


Maddox's rod-test for vertical deviation; the rod is before the right eye. *A*. The line passes through the flame—orthophoria. *B*. The line passes below the flame; the upper image belongs to the left eye—right hyperphoria. *C*. The line passes above the flame; the upper image belongs to the right eye—left hyperphoria. (DE SCHWEINITZ and RANDALL.)

eye. In esophoria we have homonymous diplopia, the streak being on the same side as the eye fixing it; in exophoria it is on the opposite side; and in hyperphoria, either below it or above it, as we have to deal with a right or left hyperphoria. Assuming that the rod be placed before the right eye, Figs. 83 and 84 show the position of the light streak and the candle flame in normally and abnormally balanced eyes.

Another well-known test is the so-called *parallax test*. This is made at the usual 6 m. distance, and is carried out with a light on a dark background, each eye being covered alternately, so as to remove the desire for binocular fixation. As the cover, placed first before one eye, is being carried before the other, the patient is asked whether the recently uncovered eye notices any movement of the distant flame. If he does not after a few trials, one may be certain that there is no marked heterophoria. When apparent motion of the candle flame is noticed, the patient will shortly be able to describe

FIG. 85.



Orthophoria. Heterophoria.

The convex spherical test.

its direction and its extent. Exophoria is indicated if the light moves in the same direction as the cover is carried from one eye to the other; if in the opposite direction, esophoria. If the light moves downward when the right eye is uncovered, there is a right hyperphoria; if it moves in an upward direction, we have to deal with a left hyperphoria. The prism that neutralizes the movement measures the degree of the heterophoria.

The Convex Spherical Test. A strong convex glass (15 D.) is covered, except at its optical centre, and placed before one eye. The distant candle image appears in the shape of a blur of light with a second image, that of an ordinary candle flame. If the muscles be normally balanced, the clear candle flame will be situated in the middle of the blurred image. In heterophoria the clear image will appear in various parts of the blurred image field, or, in the high degrees, will be separated from it. The relation between the two images and the prism required to bring the clear image into the centre of the blurred one, determines the measurement of the hetero-

phoria. Hansell and Reber believe that, when properly carried out, this test is superior to those in which prisms alone are used, and that it possesses all the advantages of the Maddox rods.

Symptoms of Heterophoria. There can be no doubt that a marked degree of almost every form of muscular imbalance (as measured by one or more phorometers) may exist without giving rise to special symptoms. The state of the nervous system, the habits of the patient, the condition of the digestive and other organs influence the symptomatology in these muscular anomalies. On the other hand, both local and general symptoms are commonly present in the majority of cases of heterophoria. Inasmuch as ametropia is intimately associated with heterophoria and with heterotropia, and since we know that the correction of the one may greatly relieve the irritation produced by the others, it is not strange that one has difficulty in differentiating the eyestrain symptoms of ametropia from those of heterophoria. We shall consider these muscular anomalies separately.

FIG. 86.

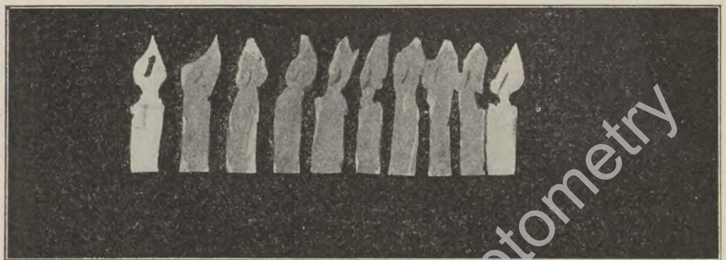


Image-movement in heterophoria, exophoria, and esophoria. (COLBURN.)

Exophoria. The tendency of the visual axes outward is generally a passive condition, and is commonly the result of loss of convergence-power or convergence-impulse, and it may range all the way from an insignificant defect to a true and almost constant exotropia. It may be due to structural defects in the insertion of the recti interni or their opponents in the too divergent orbits of wide skulls, or to some other anatomical anomaly. Moreover, an insufficient innervation of the interni may have something to do with this condition; occasionally a developmental defect in one or other internus may bring about an undue tendency to divergence. The exophoria may be paretic from the beginning, or the nerve supply having been partially restored in an exotropia, the remaining imbalance is not noticeable unless tests are applied. It will thus be seen that both exophoria and esophoria are the resultant in many cases of diseases of the most opposite character, affecting one or more of the muscles that take part in normal divergence and convergence. Anything which affects the tone of the muscular system generally, but in particular those

muscles engaged in convergence, is likely to produce an exophoria. We know both from observation and experience that the most active concomitant of exophoria is myopia. In this form of ametropia the convergence muscles are not frequently called into play; the healthy impulse to converge is usually lacking. In time loss of the convergence impulse takes place, and thus, indirectly, exophoria is produced. The symptoms caused by exophoria do not differ from those that accompany the ametropic condition and the accommodative anomalies with which it is so intimately associated. These are connected nearly always with attempts to do near work—blurring of the print, pain in and about the eyes on attempting to read or write, confusion and running together of print or of the notes in music, headache, fatigue of the eyes, and a sleepy feeling—all these may accompany a pure exophoria, even after a correction of the refractive errors that accompany it.

Treatment. We know that in many instances exophoria gives very little trouble and produces no symptoms after the correction of a simple or compound myopic astigmatism. It sometimes ceases to be an irritant when an acute or chronic disease has been cured. It is the belief of the writer that the condition of the general health and the correction of all forms of astigmatism, and especially of a concurrent myopia, should be the first consideration in this condition. de Schweinitz advises, as a routine of practice, tincture of nux vomica, fifteen drops, three times a day, increasing the dose by five daily drops until forty-five are taken or until toxic effects are noticed. When, after this has been done, exophoric symptoms still remain, attention should be directed to a permanent relief of the condition. The most important of the non-operative procedures is regular prism exercise, either by means of the Noyes-Gould apparatus or by means of ordinary square prisms set in spectacle frames and placed before the eyes. The strongest prisms, bases out, should be used. The patient fuses the double images while standing four or five feet from the point of illumination. He then slowly backs across the room, fifteen or twenty feet away. This exercise is to be performed for from three to five minutes at a time three times a day. The strength of the prisms is gradually increased, but in no case should the patient make use of such strength as to cause pain, vertigo, or other disagreeable symptoms. This calisthenic performance may also be conducted by ordering square prisms with which the patient may exercise his convergence at home. If, for example, the convergent power be 15° and the adduction 7° or 8°, three square prisms may be prescribed, of respectively 15°, 5°, and 3°. The patient uses the first for a couple of days, until fusion of the double images at twenty feet is easy when it is placed before either eye. The next day he employs, for the same purpose, the 15° and the 3° prism, and so continues until he can overcome the compound 18° prism. Finally, all three are together used, and this combination, which is about equal

to a single 25° prism, is exchanged for another series, say 25°, 10°, and 5°. In this way, the convergent power may be increased until the patient can readily overcome at least a 60° prism. Careful directions for their use should always be given, and control experiments should be made from time to time in the surgeon's office. No operation should be thought of until the measures just alluded to have been sufficiently tried. Outdoor exercise, a nutritious diet, and as little near work as possible, should always be inculcated in connection with this general treatment.

Although the constant wearing of prisms occasionally gives relief to the symptoms induced by an exophoria, the writer cannot conscientiously advise them, except as a temporary expedient. If worn for some time, the effort of convergence is more and more left in abeyance, and there is no real attempt at a cure of the conditions that

FIG. 87.

Image-movement in hypoexophoria, hyperphoria, and hypoesophoria. (COLBURN.)

underlie the exophoric state. When a prism correction is attempted, not more than two-thirds of the total amount (divided between the eyes) should be presented. Three or four degrees of exophoria rarely require any correction, and as it is usually during near work that symptoms are set up, and since the exophoria is most marked at that time, it may be advisable to give a stronger prism for reading or desk-work.

When all means fail, an operation upon the eye muscles is indicated. In this case the surgeon may tenotomize one or both externi, or advance one or both interni. The writer, while sympathizing with Landolt's preference for advancement in all cases of weakened convergence, has not been able to obtain the best results without a section (partial or complete) of the opposing external rectus. Where the apparent exophoria is really dependent upon the presence of hyperphoria or a hypertropia (as Stevens affirms is frequently the

case), no surgical interference with the exophoria is justifiable until the vertical anomaly has been corrected. Whether one or both interni require advancement, and whether accompanying sections should be done on the externi, will depend upon the results of a single operation. Probably an exophoria, symptoms of which are not relieved by glasses, medication, hygienic care, and prism exercise, should be corrected by the advancement of both internal recti, without tenotomy (partial or complete) of one or both externi. But, if the former fail, the operator need not hesitate to have recourse to the additional operations. The technique of all these operations will be considered under the allied condition—strabismus.

Esophoria. As in the case of exophoria, this condition does not give rise to distinctive symptoms. Photophobia, not explained on other grounds, blurring of the print, "panorama" vertigo and nausea, carsickness and seasickness, with headache, nervousness, and digestive disturbances, are often referred to it. As Hansell and Reber have pointed out, a curious symptom noticed by esophorics is the nervous irritation produced by the patient's seeing his nose, not only when reading or writing, but when looking in the distance. As a rule, patients suffering from esophoria are much more likely to have symptoms *due to prolonged use of their eyes in gazing at distant objects*, as, for example, in attending the theatre, going to church, in riding on elevated trains or in looking out of the window of a moving car. A small amount of esophoria may give rise in susceptible individuals to symptoms when the same or a larger amount is well borne by a patient with a stable nervous system and good digestion. The neurotic and the neurasthenic—particularly if they suffer from insomnia —are much more likely to complain of esophoria pure and simple than the healthy individual. Just as exophoria is commonly associated with myopic states, so do we find esophoria more frequently present in all degrees of hyperopia and hyperopic astigmatism.

Treatment. First of all, a full correction of any refractive error should be made, and, if necessary, glasses (usually convex) should be worn constantly, while the eyes are kept under the influence of atropine for several days or weeks. At the same time, any defect in the general health should be remedied. The habits of the patient should have attention, as these are important in dealing with this form of heterophoria. Tonics, rest from work, careful dieting, change of air, will often help to effect a cure. In the writer's experience, prism exercise has not that value which it possesses in exophoria, and yet it should be prescribed in conjunction with the foregoing treatment. Remedies having the effect of soothing the irritated nervous system are of value, and of these the most important is hyoscyamus in any of its forms. When it is impossible or not considered desirable to instil atropine, a single drop of a one- or two-grain solution of homatropine should be dropped into the eye an hour or two before bedtime. Cycloplegia produced by this agent passes off

before the next morning, while accommodative quiet is reflected in esophoric relief. Prisms for constant wear may be of occasional value, but as their tendency is to coddle the weak muscle or muscles, instead of strengthening them, this plan ought not to be encouraged. As a means of bridging over a temporary difficulty, or to allow time for improvement of the patient's health, their use may be justified, but for a permanent cure they are inadmissible. When other means fail, a marked esophoria, say, of 10°, for the distance, is a defect proper for operation. As the operative treatment of heterophoria is practically the same as that of true strabismus and other forms of heterophoria, a description of the tenotomies and advancements proper to the latter condition will be found under the appropriate heading. There is one exception to this statement, in that *partial tenotomy* and *partial shortening* are restricted, by those surgeons who employ these means, to heterophorics and to cases exhibiting minor degrees of heterotropia. The writer has occasionally had patients who were benefited by partial operations *alone*, after non-operative means had faithfully been tried.

Partial Tenotomy. This may be either central or marginal, the purpose of the former being *to lessen the tension* of the muscle operated on, while a marginal tenotomy is done both to moderate the tension and to influence the action of the muscle upon the rotation of the eyeball. Savage[1] thus describes the two operations:

The instruments needed are the same as those required in doing the complete operation. To do a central, partial tenotomy the lids must be well separated by the speculum. The patient should look as far as possible in the direction opposite the muscle to be operated on. The conjunctiva over the insertion of the tendon should be lifted in a meridianal fold with the forceps, and this should be snipped with the scissors. Through the cut in the conjunctiva the forceps should be made to grasp the capsule of Tenon, which in turn should be snipped through the opening in the conjunctiva; the central fibres of the tendon should then be grasped with the forceps and slightly raised from the sclera, so that they may be caught with the scissors between the forceps and the attachment, as close to the latter as possible. Thus the tendon is buttonholed. If the operator is certain, from the resistance he feels with the forceps, that he is not too near either margin of the tendon, he may divide a few more fibres in both directions, while still holding the tendons with the forceps; but in doing so he takes some risk of doing too much. Now the forceps should be laid down for the small (Stevens) hook, which should be passed through the buttonhole in the tendon, first in one direction, then in the other, beneath the uncut fibres, so as to determine the resistance. Guided by the hook, the operator now divides fibre after fibre with the scissors, until the lessened resistance warns him that he has gone far enough in that direction; he then repeats this step

[1] From the advance sheets of "Ophthalmic Myology."

toward the other margin, in the same careful way. To get the full effect of a partial tenotomy, the capsule of Tenon must be cut coextensively with the division of the tendon. The cut in the conjunctiva may or may not be of the same extent. There is no necessity for making either a very small or a very large conjunctival incision; but for those just beginning, a large conjunctival incision would make the tenotomy both easier and safer. In a *marginal tenotomy* the initial cut of the conjunctiva, capsule, and tendon is made as for a central tenotomy, care being exercised that the buttonhole in the tendon, if not in the centre, shall be nearer that margin which is to be completely severed later. Still holding the tendon with the forceps, the scissors may be passed in the direction in which complete division is indicated, and be made to cut all the fibres at once.

Hyperphoria. In this anomaly there is a tendency of one visual line to project itself higher or lower than that of the other. Hyperphoria is by no means a rare condition, and, although it may be present to a marked extent, it does not always excite symptoms, particularly if the individual possess a sound nervous system, a good digestion, and does not abuse his eyes. The principal symptom, not only in hyperphoria, but to a greater or less extent in all the muscular anomalies, is, in the writer's experience, *sensitiveness to light.* In the case of hyperphoria we find also the usual reflex symptoms: hyperæmia of the lids giving rise to smarting, burning, and a sensation of heat in the eye; confusion of images, particularly when the patient is walking along a crowded street, looking out of the window of a car in motion, ascending in an elevator, etc. It happens not infrequently that hyperphoria of slight degree produces more ocular and other (nervous) symptoms than one would expect. Not only in this form of heterophoria, but in all the others, the symptoms are not in direct proportion to the amount of the defect; indeed, just as it is impossible to say how much hypermetropia or astigmatism is required for the production of symptoms in a particular individual, so is it difficult to indicate the amount or kind of annoyance likely to accompany a given amount of hyperphoria in a certain patient. This defect gives rise, not only to what Bennett has termed "panorama" symptoms—as shown by headaches, vertigo, and occasionally vertical diplopia—but also evidences of nervous irritation on attempting to use the eyes for long-continued near work are rarely absent. These are, in particular, dizziness, ocular pain, and photophobia. The patient is given to squinting his eyes during both near and distant fixation; furrows or ridges may be usually detected above one or both eyebrows, or the eyes may present a staring appearance, or there may seem to be an apparent ptosis of one lid, with a wide-open condition of the other. This peculiar wrinkling of the brow is seen in its exaggerated form in true oculomuscular paresis, but its meaning is the same in hyperphoria. The head, also, is very often carried with a tilt away from the hyperphoric eye. If the vertical defect be not

excessive, this carriage of the head may be sufficient to overcome the whole of the hyperphoria, and it is a common experience that many hyperphorics, physically well developed, succeed during their ordinary occupations in obtaining comfort by thus neutralizing all, or nearly all, of an otherwise intolerable hyperphoria. Exophoria is very frequently associated with hyperphoria, and, as Stevens points out, correction of the one may issue in cure of the other. Indeed, the writer is convinced, from an examination of a large number of these cases, that hyperexophoria and hyperesophoria are often results of an attempt on the part of the lateral muscles to relieve the vertical deflection. This point should always be decided before an attempt is made to correct either anomaly. Both supraduction and infraduction, as well as abduction and adduction, should be carefully measured with and without correcting-glasses. If the lateral deviations are found to be in normal relation to one another, and the vertical excursions are abnormal, one may conclude that the case is essentially one of hyperphoria.

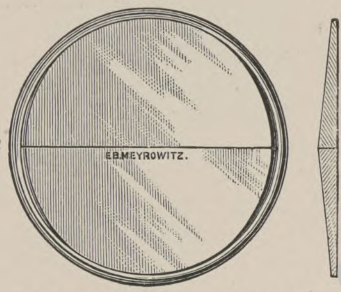
Tests for Hyperphoria. These have already been referred to, but it is well to say, in addition, that since small degrees of this defect are of greater importance than minor amounts of exophoria or esophoria, care should be exercised in eliciting its presence or absence. The parallax or the screen test will be found of particular value in the detection of this anomaly. According to Duane, constant practice will enable one to detect as little as a quarter of a degree of hyperphoria.

Treatment. First of all, there should be a thorough correction, under a cycloplegic (when that is necessary), of all refractive errors. This will be found sufficient, in many cases, to make the patient so comfortable that further interference, for the time at least, is uncalled for. If correcting lenses are found to be insufficient, prisms should be worn. It must be confessed that it is not easy in the first instance to say what proportion of the deviation should be represented by prisms. As a rule, most patients will not tolerate more than one-half the full correction, divided between the two eyes. The writer's experience is that it is best to order the prism compounded with the correcting glass, and he does not find that fronts are comfortably worn. It is better to have two pairs of glasses if it is decided to use different prismatic strengths for distant and near work. The power of infraduction and supraduction, as measured from time to time, should decide whether the prisms thus constantly worn are to be decreased or increased. The constant wearing of prisms is of greater benefit and more justifiable in hyperphoric conditions than in those of esophoria or exophoria, and they often produce brilliant results. Savage advises exercise with vertical prisms, after the manner suggested for prism-training in weakened convergence and divergence, but the results are not satisfactory.

The same rules apply in the operative treatment of hyperphoria

that were laid down for esophoria and exophoria. After all other plans (correction of ametropia, attention to general health, etc.) have failed, tenotomy of the overstrong, or rather overworked, muscle is indicated. Care should be observed not to operate in paretic cases. When in doubt the tropometer or any of the other means of measuring the rotating power of the individual muscles will be found of great value. It is not always easy (owing to the secondary contractions following even a slight paresis of a vertical muscle) to decide whether we have to deal with a non-paretic hyperphoria or not. When the latter is constant in amount, and is found to be due to insufficient power of one of the vertical muscles, the proper procedure is an advancement or a muscle-shortening; where the defect is due to overaction, tenotomy is indicated. If there be a field for partial tenotomies, it is in low degrees of hyperphoria due to overaction of a particular muscle. But the same objection to the tenotomy of a muscle is as pertinent in hyperphoria as in other muscular anomalies; it is always better to strengthen a weak muscle than to weaken a strong one, even when balance of all the muscles is the object sought.

Fig. 88.



Maddox double prism. *a*. Front view. *b*. Sectional view.

Cyclophoria. This is an insufficiency of the oblique muscles, or a defect of the normal torsion or wheel-motion, which occurs when the oblique muscles especially are called into action. We are indebted for most that we know of this subject to Savage. He attributes a number of symptoms (common in ametropia and other forms of heterophoria) to this condition. It may be detected by covering one eye and placing over the other a Maddox double prism.

The patient fixes a horizontal line drawn on a white card eighteen inches from his face. He sees two lines. The second eye, the one being tested, is now uncovered and a third line will be seen halfway between and parallel to the others, if all the muscles are properly balanced. In imbalance of the oblique muscles the central line is tilted, in relation to the others, either up or down. If the middle line be nearer the bottom than the top line, or *vice versa*, there is a hyperphoria; or, if the middle line extend more to the right or to the left, abduction or adduction is at fault.

Treatment. Rhythmic exercise of the insufficient oblique muscles is accomplished by the method of Savage. This consists of the rotation of convex or concave cylinders before the eyes of the patient who, meantime, looks at a distant candle flame. We have had so little experience of the results of operations upon the obliques for the relief of cyclophoria, that it can only be said that the matter is still *sub judice*.

Strabismus. **Squint.** **Heterotropia.**[1] In this condition the visual axes are so directed that the image of the object does not fall upon the fovea of both eyes at the same time. There is an absence of binocular vision, although the rotating power of the individual muscles is not to any great extent impaired.

Internal or Convergent Strabismus. CONVERGENT SQUINT. ESOTROPIA. The visual axis of one eye is directed toward that of its fellow, so that the image falls upon some portion of the retina in the deviating or squinting eye outside the fovea. Convergent strabismus may be monocular or constant, binocular or alternating. In the former case, one eye is constantly used for purposes of fixation, while the second eye turns in. In the second case either eye is used indifferently for fixation, and the opposite eye squints.

There is practically no definite line of demarcation between squint and heterophoria. Some forms of heterophoria may represent an early stage of strabismus, or the same muscular imbalance may at one time be properly called squint, and at another time a mere insufficiency of the muscles.

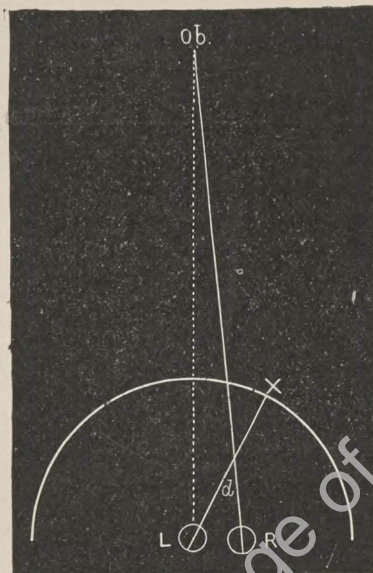
Monocular Squint or Constant Squint. Not only is the vision of the strabismic eye defective, but the amblyopia generally does not correspond to any ophthalmoscopic defect, although the visual field frequently is contracted. The error of refraction of the amblyopic is often much the same as that of the fixing eye, so that the ametropia alone cannot account for the lowered visual acuity. Probably there is a true *amblyopia ex anopsia;* although in the majority of cases the vision of the non-fixing eye is not to any extent improved by correction of the refractive error and cure of the squint. Whatever be the origin of the defect in sight, it is probably the main cause of the heterotropia. Without discussing the various theories from time to time put forward to explain squinting eyes, one may say that the brain centres have a dislike for the vision that results from an eye that sees plainly and one that sees indistinctly, and that in the effort to rid the nervous system of this source of irritation the defective eye is turned in the direction and kept in the position easiest to obtain and maintain. If, however, we are enabled by any means to improve the vision of the squinting eye, we to the same extent remove the objection on the part of the nervous system to eyes of unequal vision. If, in addition to this desirable result, there goes a restoration to parallelism of the visual axes, we may obtain not only binocular sight, but also comfortable vision with both eyes.

Diagnosis. Inspection of the eyes will generally show an abnormal direction of the visual axes, and thé sclera will be found more exposed in one segment than in the corresponding portion of the opposite eye. The cornea will, in most cases, be seen to be deflected toward the

[1] Some authorities prefer the expression *heterotropia* for all forms of imbalance of the muscles in which binocular fixation is lost, discarding the terms "strabismus," "paralytic squint," etc. Doubtless, in time, this nomenclature will prevail.

nose. This apparent deviation of the visual axes is not always to be relied upon; the angle alpha may be abnormally small, so that although there is no muscular imbalance, there is an apparent esotropia. The best means of diagnosis in cases of doubt is the cover test. If the fixing eye be excluded by a screen, the cornea of the deviating eye will be seen to make an excursion outward. The eye that before turned toward the nose, now attempts to fix, and in doing so the cornea is rotated so that the rays of light may pass through and fall upon the foveal region. Owing to the high grade of amblyopia in some eyes, it is not easy to measure the amount of squint with prisms, and for the same reason the use of the double images of a diplopia in the various phorometers usually fails to furnish any information. *The false image is suppressed in most cases of convergent and other forms of squint.* The amount of excessive convergence in squint may, according to the method of Landolt, be measured on the perimeter. The patient fixes with the better eye while a cand e is carried along the arm of the perimeter until its reflection is seen by the observer from the centre of the pupillary area of the opposite cornea. The angle thus subtended is read off on the arc of the perimeter. (Fig. 89.)

FIG. 89.



Measurement of squint with a perimeter. (LANDOLT.)

Treatment. Since convergent strabismus usually is associated with, and is by some said to depend in most cases upon the presence of hypermetropia (as an indirect result of abnormal accommodative effort, we have increased attempts at convergence), paralyzing the accommodation with an effective cycloplegic, like atropine, is indicated. At first the squint is usually relieved, or it may not undergo any sensible diminution for some days or weeks. When the patient's accommodation is thus thoroughly paralyzed, a full correction of the hyperopic error should be ordered and worn constantly. The effect of glasses may be much increased by the continued use of atropine. The writer's plan is to instil a single drop of a 1 per cent. solution of atropine sulphate into each eye after breakfast; this is continued for two weeks after the glasses have been ordered. The cycloplegic is then stopped for two weeks, or until the patient's pupils are no longer dilated. If the glasses are not fully accepted, or if there is no improvement in the squint, the atropine cycloplegia is

continued for another fortnight, and so on alternately every fortnight for from three to six months, during which time additional attempts should be made to educate the faculty of fusion and to promote binocular vision both for distance and near. One of the best means of accomplishing these important results is the use of the *stereoscope*, using with it, for example, the pictures of Kroll. The method of Landolt, in which the vision of the better eye is dulled, so that it more closely approaches that of the squinting eye, is the one preferred by the writer, and, although much patience will be required, both on the part of the surgeon and of the child, good results are often obtainable by the use of this simple instrument. As convergent strabismus usually sets in during childhood, several questions arise in connection with this fact. In the first place, how early shall we attempt medication? The answer is that atropine should be used as soon as the squint appears and attempts at monocular fixation are made. The writer has frequently begun to treat a convergent strabismus in children two years of age, and has had them wearing glasses with benefit before they were three years old. If we should fail in obtaining parallelism of the eye, or should so far succeed in our efforts as to convert the monocular squint into one of occasional esotropia, an operation should be done—the earlier the better. In monocular squint that has defied milder measures, advancement of the externus with tenotomy of the internal rectus is the operation that will generally be required and that will usually be successful. One often finds an upward deviation combined with the inward squint, and it is usually necessary in such cases to tenotomize also the superior rectus of the squinting eye.

Alternating Convergent Strabismus. In this form of convergent squint the nerve centres seem indifferent as to whether vision is conducted by one eye or the other; sometimes one eye fixes and the other squints; sometimes the hitherto squinting eye sees, while the fellow eye turns in. Vision is usually about the same in each eye, and it is usually easier to restore binocular vision than in constant squint. It is sometimes difficult to explain the origin of alternating strabismus, especially examples of it where there seems to be an objection on the part of the cerebral centres to binocular fusion. One may at least postulate a congenital defect of co-ordination in the fusion centres. These patients invariably have a hypermetropia of more than 2 D., and we may assume that the incentive to accommodative effort and the abnormal use of the convergence have something to do with the squint.

Diagnosis. This is practically the same as for the constant form. It is easier, however, to induce diplopia with prisms or otherwise, and so recognize the relation of the false to the true image. This is, of course, much more difficult in children, but with a little coaxing and the exercise of patience, it can often be demonstrated. Hyperesotropia is a common variety of alternating squint, just as it is in

the constant form, and in the procedures undertaken for its cure this form of the defect should not be overlooked. The treatment of alternating esotropia is practically that of the constant variety, and, although the writer does not entertain the enthusiastic opinion commonly held as to the high proportion of cures in binocular strabismus convergens, there is no doubt that parallelism and binocular fusion more frequently result from judicious treatment than they do in the constant form.

External or Divergent Squint; Divergent Strabismus; Exotropia. This is the antipodes of esotropia, and is usually associated with myopic eyes. It is an acquired condition, often dependent upon the same causes that bring about myopia, such as asymmetrical orbits, long-continued near work, insufficiency of the internal recti, etc. The etiology of many cases is obscure, but the lessened demand on accommodation and convergence, and the consequent relaxation of the interni muscles that accompanies the acquisition of axial myopia, are largely responsible for them. The eye turns out constantly in the monocular form, and alternates in this position with the fellow eye in binocular exotropia. Probably the disease begins with a tendency to deviation (exophoria) and ends in a true exotropia. For this reason divergent squint is rarely seen in children, but is an anomaly of adolescence. The tendency of myopic eyes toward divergence is partly due to the enlargement and consequent elongation of the eyeball, which adds to the weakness of the adducting muscles. This train of causes, with the decreasing convergent power, brings about a true divergence. Small degrees of divergence are readily detected by the cover test, while a high degree is readily seen on inspection. Occasionally the squinting eye in constant exotropia is amblyopic, but the deviation itself rarely sets up symptoms, because the image of the divergent eye is suppressed and diplopia is not a symptom.

Treatment. The optical treatment of divergent strabismus consists chiefly in the correction of the accompanying myopia, with or without the prescription of prisms. As full a correction of the myopia as the patient will tolerate should be given, and the accommodation should be further stimulated by instilling a weak solution of pilocarpine into each eye three times a day. If the exotropia be associated with hypermetropia, the latter should either not be corrected at all, or the weakest working-glass should be used. In this way one may hope to excite the action of the ciliary muscle and arouse a concomitant stimulation of the adductor muscles, and thus lessen the divergence. In low degrees of exotropia, or in those cases where the aforementioned treatment is successful in converting the exotropia into an exophoria, prism and stereoscope training should be made use of.

After a fair trial has been given these remedies, and parallelism or binocular fixation is not attained, operative measures are called for. The remaining deviation should be overcome by advancement of the internal rectus of one or both eyes, not forgetting the

vertical deviations that sometimes accompany this form of squint. When the patient has once possessed the power of binocular vision, the operative treatment is much more likely to be successful, although a period of stereoscope training may properly occupy several months both before and after operation.

Hypertropia. Vertical deviation amounting to squint is usually associated, as has been mentioned, with exophoria or esophoria; in any event it may be regarded as a late stage or exaggerated form of hyperphoria. Many patients exhibit a deviation in the vertical line which may at one moment present a hyperphoria and at another a hypertropia. As long as the patient's fusion power is sufficient at any time to bring about binocular vision, the former condition obtains; the moment this fails one eye turns up, and we have a vertical squint. What has been said of hypophoria is largely true of hypertropia, both as to causation and treatment.

Anaphoria, Anatropia. Cataphoria, Catatropia. Stevens was the first to describe a class of cases in which both visual axes deviate either above or below the horizontal plane. If the condition be pronounced and require careful testing to prove its existence, he designates the anomaly *anaphoria,* when there is a deviation of both axes upward, or *cataphoria* in deviation of both axes downward. If the imbalance be more marked, we have an anatropia, or a catatropia. Whatever be the nature of this unusual condition, it is discovered by means of the screen or cover test. In anatropia the right eye turns up behind the screen, while the left eye fixes, the left eye rotating upward, and not downward as in hypertropia, the moment the cover is transferred to the other eye. In catatropia a downward excursion is noted in both eyes with the cover test. Stevens attributes a number of evil consequences to these conditions, which he removes by operation.

Paralysis of the Eye Muscles. Although for the purpose of investigation it is desirable to consider the ocular palsies apart from heterotropia and the various forms of squint, it is often difficult to differentiate one from the other. Just where functional weakness of a muscle ends and paretic insufficiency begins is, upon occasions, impossible to demonstrate. Muscular paralysis may be of intracranial or orbital origin; in other words, the nerves and nerve centres upon which their function depends may be attacked in the cortex (cortical paralysis), in both cortex and nuclei (cortico-nuclear), in the nuclei alone (nuclear), within the cranium and along the periphery (cranio-peripheral), or within the orbit (orbital). The central causes of these organic lesions are commonly constitutional, especially lues, rheumatism, and tuberculosis. They are often associated with tabes, paralysis of the insane, brain tumors, basilar meningitis, and other processes inherent in the various forms of intoxication, tuberculosis, diphtheria, hysteria, nephritis, diabetes, direct and indirect injury, etc. Of congenital paralysis, *ptosis* is the most common sign, while paresis of the external rectus alone is frequently encountered.

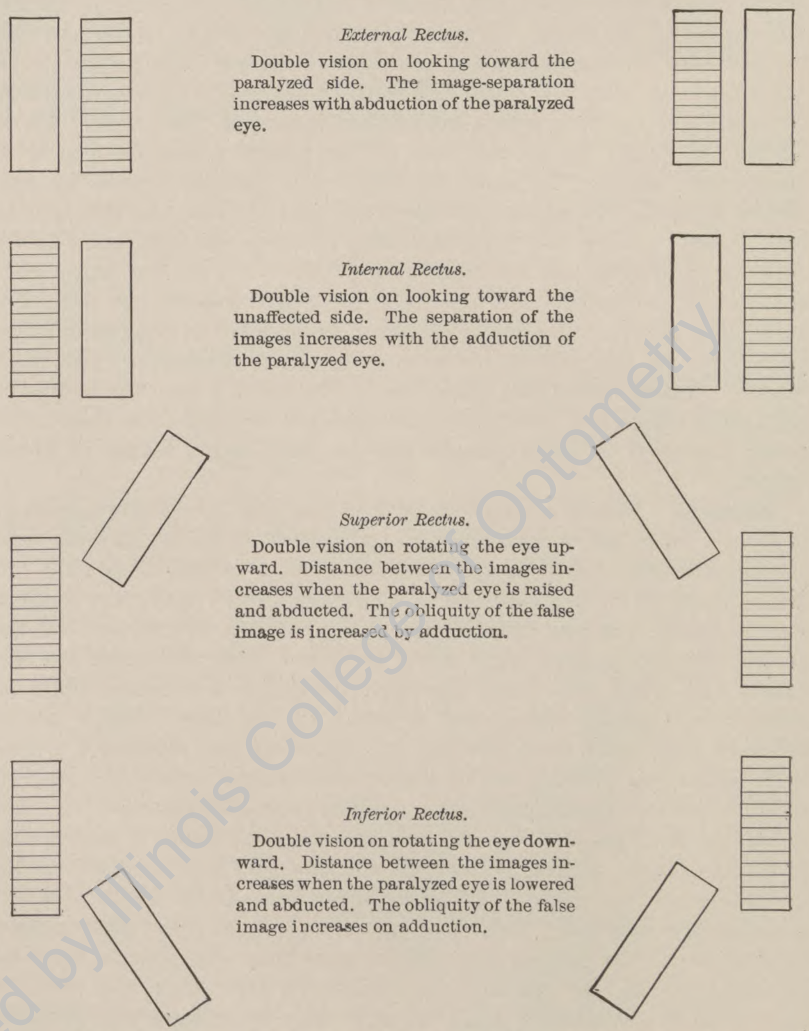
Symptoms. Ocular paralysis sets in without warning, unless it be headache, or the symptoms of disease (generally of the nervous system), or traumatism with which it is so often associated. *Double vision* is usually the first indication the patient has of an attack. It is extremely annoying, particularly if it be oblique or vertical. The diplopia persists during the attack, and is often associated with *vertigo, nausea, occasional vomiting, mental confusion, uncertain gait,* and a *sense of insecurity* while walking about. The eye under the influence of paretic muscles does not see objects in their proper position, and to this *false projection* are due the uncertainty in walking and grasping objects and other disagreeable consequences of the paralysis. The usual innervation effort put forth to assist the disabled eye to fix is the source of the error. It was employed by Graefe under the name "touch test" in testing for the paralyzed muscles. The patient covers the sound eye with one hand and with his forefinger endeavors quickly to touch the tip of a pencil held before him with the other. He will direct the finger tip to the side of the pencil corresponding to the paralyzed muscle. In time, however, the patient learns by experience to make allowance for this error, and for him the test is valueless. After a time the patient learns to close one eye, so as to shut out the confusing second image; or if ptosis set in, the same end is served. Eventually, also, the head is turned toward the side of the paralyzed muscle (upward in elevator, downward in depressor muscle paralysis), as this action corrects or at least diminishes the double vision. These unnatural positions of the head assist the surgeon in making a diagnosis, although it should not be forgotten that similar poses are seen in heterophoria and in some other forms of heterotropia.

Diagnosis. When a single muscle in one eye is recently paralyzed, it is easy from the symptoms and by inspection to say at once where the trouble lies. More frequently, however, a careful inspection of all the excursions of both globes is necessary before a correct diagnosis can be made. In any event the patient should be placed with his head in the primary position and asked to follow, first with each eye separately and then with both together, the point of a pencil while it is carried in various directions in front of his face. Many are the schemes that have been devised for detecting the character of paralytic squint by making use of the double images invariably produced in some part of the field by even a slight muscular paresis. Of these, probably Mauthner's (Fig. 90) pictorial table of single muscle paresis is the simplest. There are several reasons why it is so often difficult to determine by means of the diplopia test alone what muscle or muscles are affected. One does not always have intelligent patients to deal with; the paresis may affect more than one muscle, or it may be complete in one eye and incomplete in the other; or, when one eye alone is affected, a single muscle may be completely paralyzed while others are only partially affected. Moreover, when the case is

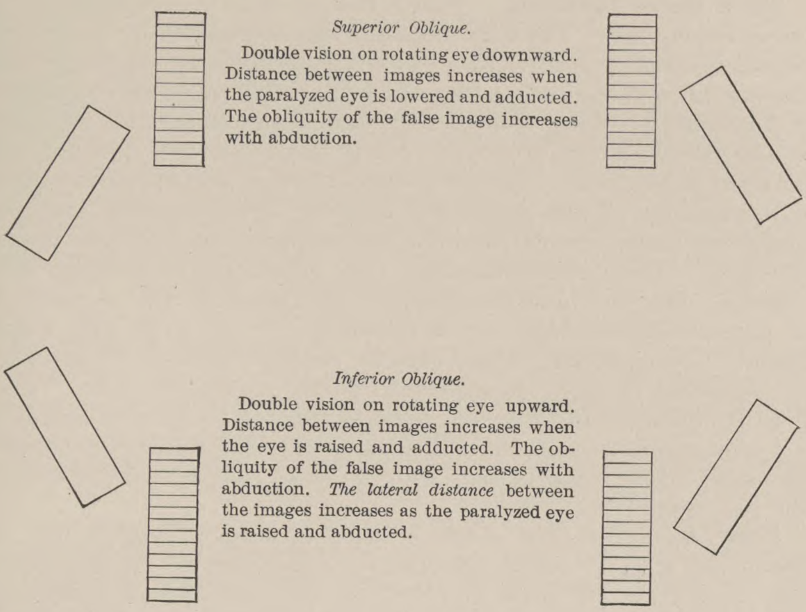
of long standing, contractures of the antagonist muscles are pretty sure to set in, and there may also be in such cases a successful effort, made in the interests of the nervous system, to suppress entirely the image in the deflected eye. A previously existing muscle imbalance or a marked difference in the vision of the two eyes also acts as a disturbing factor. There may be no squint when the eyes are turned away from the paralyzed muscle, but it is readily produced when they

FIG. 90.

RELATIONS OF THE DOUBLE IMAGES IN PARALYSIS OF THE OCULAR MUSCLES.
(The True Image is Barred.)



Mauthner's scheme for the detection of the affected muscles in ocular palsy.



Mauthner's scheme for the detection of the affected muscles in ocular palsy.

are rotated toward the seat of paralysis. The deviation is more marked the wider the attempted excursion, while the limitation of movement noticed can usually be referred, without difficulty, to the proper muscle or set of muscles. *If the affected eye fix an object directly in front of it and the sound eye be covered, the latter will deviate to a greater extent in the same direction than the paralyzed eye.* This *secondary* squint is an overaction result arising from the excessive innervation effort needed to allow the affected eye to fix. This fact is to be remembered in the differential diagnosis between functional and organic esotropia; in the former the primary and secondary deviations are equal.

Unilateral Paralyses of the Orbital Muscles. Paralysis of the External Rectus. Abducens Paresis. Paralysis of the Sixth Nerve. This is the commonest form of the individual palsies. The long course of the sixth nerve through its bony canals renders it peculiarly liable to disease from the various meningitic and other processes that may occur during its passage to the external rectus muscle. It is often found as a part of rheumatic, syphilitic, and traumatic conditions, as well as in disease of those central neurons with which the sixth nerve is associated. Wood says that when the paralysis is peripheral it is likely to be due in adults to syphilis when not distinctly of rheumatic origin, but that it is generally tubercular in children.

Third Nerve Paralysis. This is next in order of frequency of the one-sided pareses. The most common sign is paralysis of the levator palpebræ, causing *ptosis*, with a loss of the normal skin-wrinkling of the affected lid, although the latter can be partially raised by contraction of the frontalis muscle. When other branches are implicated, the globe is defective in all its excursions except the downward, upward, and outward movements. The eye deviates outward and downward owing to contraction of the unaffected superior oblique muscle, and the upper end of the vertical meridian will be plainly seen to turn toward the nose. There is *exophthalmos* from relaxation of so many recti muscles; dilatation and immobility of the pupil, as well as paralysis of accommodation from the involvement of the iridic and ciliary fibres. The *mydriasis*, which may be further increased by atropine, is unaffected by light, convergence, or the consensual test. Vision both for distance and the near point is affected about as it would be if a cycloplegic were instilled into the eye. The diplopia is crossed, the false image being higher, and its upper end is inclined toward the paralyzed side.

Paralysis of the third nerve is often incomplete, and it may be associated with the same affection of other nerves. If the ciliary muscle and iris are alone involved, we have an *internal ophthalmoplegia;* if the extrinsic muscles are all affected, an *external ophthalmoplegia;* if both external and internal muscles are paralyzed, a *total ophthalmoplegia.*

A form of recurrent oculomotor paresis, called by Charcot *ophthalmoplegic migraine*, attacks children and young adults who suffer from severe headache (attended by nausea and vomiting) on the side of the paralyzed muscles. In the intervals of the early attacks, which last from a few days to a few months, the muscles regain their normal functions, but the paresis becomes more marked and at last it may be permanent. The disease affects both sexes equally, and is accompanied by contraction of the field of vision and lowering of the central acuity. Its real nature is obscure; some writers believe it to be hysterical, others attribute the symptoms to a lesion of the nerve root at the base of the brain. No treatment is of avail.

Paralysis of the Superior Rectus. This is not an uncommon unilateral paralysis. With it there is limited movement upward and toward the unaffected eye, accompanied by diplopia in the upper half of the field of vision. When the patient looks in this direction divergence is the result. The face, in fixation, is turned up, while both it and the head are inclined toward the sound side. The relative position of the true and false images will be seen on consulting the chart (page 178).

Paralysis of the Superior Oblique. Trochlearis Palsy. Fourth Nerve Paralysis. This muscle is rarely paralyzed alone. The diagnosis can usually be made in recent cases by the diplopia-scheme test (page 179), or by remembering that there is homonymous diplopia on

looking down, that the false image is lower, with its upper end inclined toward the healthy eye. It is a very troublesome form of paralysis, and the patient is compelled to close one eye to avoid the double vision in the lower half of the field.

Unilateral and isolated paralysis of the inferior rectus, internal rectus, or inferior oblique is extremely rare. When any of these does occur, it can, especially in recent cases, be diagnosed by the symptoms and a study of the positions and relations of the diplopic images.

A fairly large percentage of ocular palsies affect the associated movements of the two eyes, and while, as before stated, almost any or every combination of paralysis of the muscles of the two eyes may occur, there are particular examples that call for mention.

Paralysis of Convergence. This may result from true nuclear or supranuclear disease. It is not necessarily followed by diplopia, but the patient is unable to fix with either eye at the near point; the optic axes remain parallel in all movements.

Conjugate Paralysis. Inability to move both eyes together, either to the right or to the left, while the convergent power is preserved, is not infrequently seen. The lesion in this case is probably cortical, although it is also claimed to be near the sixth nerve nucleus—said by some observers to be the centre for the associated lateral motions of the globe. It is often a distant symptom, as in hemorrhage into or disease of the cortex, pons, internal capsule, etc. It usually lasts but a short time, because disturbance of the centre in one side of the brain is soon quelled by the unaffected second centre. *In destructive lesions* with this symptom the eyes turn from the paralyzed side (Swanzy) when the cerebrum is the seat of the disease, but toward the paralytic side in pontine disease; the eyes turn toward the convulsed side *in irritative lesions* of the cerebrum, but away from it in irritation of the pons.

Conjugate paralysis of both upward and downward movement, due to disease of the thalamus opticus and the corpus striatum, has also been recorded.

Prognosis. As a rule, the peripheral paralysis (due to exposure to cold, rheumatism, injury) gets well, but where the cause of the palsy is intracranial the prospect of a cure is necessarily more remote. The first attack of ocular paresis that heralds the approach or forms a part of tabes dorsalis may disappear, only to recur and become permanent. Indeed, it may well be remembered that an attack of ocular paresis occurring in a man over thirty-five years of age—particularly if he has had early syphilis—should arouse suspicions of a probable posterior spinal sclerosis. Probably the paretic cerebral complications of syphilis (gumma, local periostitis and the like) are as amenable to treatment as any paralysis of central origin. In most cases many weeks or months may elapse before improvement or cure results. The longer a paralysis has existed (with or without treatment) the less the hope of eventual cure.

Treatment. So far as possible the cause of the paralysis should be removed. Where the origin of the trouble is definitely rheumatic, salicin, ten to twenty grains three times daily, has, in the writer's hands, been found more desirable and more readily borne than the salicylates. This should be combined with an antirheumatic regimen, vapor baths, and copious draughts of lithia water. Injuries should have the care proper to them. All the other cases, unless there is some contraindication, should at once be ordered a course of sodic or potassic iodide (or both together) in increasing doses, to be given between meals and in a large quantity (pint or two) of water. Most patients will tolerate 300 or 400 grains daily with benefit. This may be supplemented by mild mercurial inunctions, and the patient should meantime take, three or four times every week, the Turkish or the ordinary sweat bath. For the benefit of the change (as well as the hot water) patients do well at various hot springs here and abroad. Coincident with this, the treatment proper to gout, arterio-sclerosis, tubercle, and brain neoplasms is indicated, although in many instances it will not be followed by any good result so far as the paralysis is concerned.

Locally, a weak current (2 to 5 milliampères) of the interrupted galvanic current will be found of use—the cathode over the closed lid or on the cocainized sclera, near the insertion of the paralyzed muscle, the anode at the nape of the neck.

Michel's plan of grasping the cocainized conjunctiva and sclera with a pair of fixation forceps, and forcibly exercising the enfeebled muscle by rotating the globe back and forth in the direction of its action about a minute each day, is of some value. Other forms of exercise, with prisms or fixing a near object for a few minutes at a time several times a day in all possible directions, may have the effect of preventing secondary contractures and of stimulating the peripheral nerve fibres. Sometimes, when the paresis is slight, correcting prisms relieve the diplopia and the vertigo. In chronic paralysis, after the foregoing treatment has been applied without success, when the paretic muscle retains some contractile power, advancement of the weak muscle with Tenon's capsule may be tried. Section of the antagonist will always be needed. If the paralysis be complete, no operation should be undertaken.

Paralysis of the Extrinsic Muscles in the Localization of Cerebral Diseases. Third Nerve Paralysis. Bearing in mind the nuclear and cortical centres of the eye muscles, *unilateral ptosis* alone is nearly always due to implication of the cortical (associated) centre in the opposite upper extremity of the ascending frontal convolution—near the arm centre. Isolated ptosis is, hence, called *cerebral ptosis.* Paresis of the levator palpebræ occurring *on the same side as the lesion,* without implication of the other branches of the third nerve, indicates disease of the pons Varolii. In destructive lesions of the crus where there is *crossed* paralysis, ptosis is usually present as part of a total

third nerve paralysis. If, under these circumstances, only the branch supplied to the levator be affected, one may diagnosticate a lesion of the peduncle. When oculomotor paresis is found on the same side and about the same time as a central lesion shows itself, with loss of sensation and motion (including facial and sometimes hypoglossal paralysis) of the *opposite* side of the body, we have a "crossed hemiplegia" that almost invariably means destructive disease of the crus. Lesions affecting the basal neurons are, however, the commonest of the oculomotor paralyses, and these are usually complete. It is not always easy, from the character of the paralysis alone, to differentiate between disease of the crus and purely basal disease. If there is no other paresis, or if there is an incomplete hemiplegia with the third nerve paralysis, the changes are almost certainly at the base of the brain. One must not forget that oculomotor paralysis may occur as a distant (pressure) symptom, especially in brain tumor and thrombosis of the cavernous sinus.

Paralysis of the Sixth Nerve. Owing to the many connections formed by the abducens during its long course from the brain to the external rectus muscle, it is subject to paralysis in lesions not directly reaching its nuclear origin. Cerebellar tumor is an example of a distant lesion especially prone to affect the sixth nerve in this way, and one or both nerves may be compromised. When abducens paralysis appears as the *only* focal sign, it usually means basal disease, and, apart from fracture of the petrous portion of the temporal bone, is likely to be due to syphilis, particularly if it be bilateral. When paralysis sets in with an *opposite* hemiplegia and other evidence of cerebral disease, the lesion can be referred with confidence to the pons. A hemiplegia due to a lesion in the cortical motor area furnishes much the same symptoms, except that the paralysis is on the same side. Owing to the close relations of the nuclear centres for the sixth and seventh nerves we often have facial and abducens paralysis occurring together. When these are associated with a crossed hemiplegia the lesion is in the pons.

Paralysis of the Fourth Nerve alone is a very rare occurrence in cerebral disease; when associated with paralysis of other oculomotor nerves it is practically impossible to separate it as a localizing sign. In the former case it is the result of a basal lesion; when it sets in with third nerve paralysis it indicates a lesion of the peduncle.

As Prevost has pointed out, and as we have just seen, in *conjugate* deviation of the globe due to paralysis of the associated muscles the eyes are turned toward the side upon which the central lesion is situated.

Spasm of Accommodation. The constant demands made upon the ciliary muscle and the habit so engendered are such that, in young persons especially, relaxation of the muscular contractions does not always take place, so that the true state of the refraction is masked. If the patient be hyperopic, he may appear emmetropic or myopic; if

emmetropic myopic, and if myopic the myopia may seem to be greater than it really is. This fact furnishes the reason why a patient may present perfect distant vision as measured by test-types, and yet have a fairly high degree of hyperopia or astigmatism, or both. In other cases the spasm is so marked that a hyperope may have greatly diminished distant vision and appear to be myopic three, four, or five dioptres. Concave lenses may, in such instances, be accepted and apparently restore the lost vision for a time, but visual acts will be painful, and all the other signs of eyestrain (headache especially) are likely to be present. The occurrence of accommodative spasm teaches us the need of paralyzing the ciliary muscle with atropine, or some other cycloplegic, before measuring the refraction; otherwise we cannot be certain of the condition we have to deal with, unless the patient be past forty years of age. Moreover, it is wise to assist in breaking off the spastic habit by ordering the patient to wear the glasses before the effects of the cycloplegic have passed away.

Spasmodic or Spastic Heterophoria. Just as spasm of the accommodation occasionally arises from strain of the ciliary muscle, so may we have overaction and cramp of the straight muscles. In their efforts to overcome a muscular imbalance some particular muscle may be so stimulated to overwork that the real nature of the heterophoria is completely masked. This is the reason why a complete correction of the refractive error should be made, preceded or followed by muscular rest, before dealing with the heterophoric defect. It often happens that an apparent heterophoria disappears and the patient is made comfortable after glasses are ordered. The relief afforded the ciliary muscle is reflected upon the tasks of the orbital muscles. For example, an apparent esophoria for near may become an orthophoria to tests when convex working-glasses are employed; or a right hyperphoria may dissolve into a left-sided vertical defect after the use of prisms, or of lenses correcting the ametropia, or from the employment of both.

Certain forms of manifest hypoexophoria and hypoesophoria are either pure hyperphorias or pure horizontal deflections, the impulse for binocular vision so affecting the related muscles that they come to the aid of the defective ones and bear most of the burden. It behooves the surgeon, therefore, to make a number of tests at intervals before deciding in doubtful cases, and, if possible, to keep the patient for a week or more under the influence of a cycloplegic. When persistent contraction of a muscle has lasted for months or years a form of tonic cramp arises that may require, in addition to these measures, tenotomy of its tendon with or without shortening or advancement of the opposing muscle.

Operations on the Eye Muscles. When milder means are found insufficient to restore binocular fixation or to relieve spastic strain, operative interference is indicated in most cases. The chief point

to be borne in mind is the need of conserving the rotating force of the ocular muscles. It is consequently better for the future of the patient to strengthen a weak muscle in our attempts to bring about the necessary balance of power than to accomplish it by reducing the effectiveness of the stronger muscle, even if we know that its overaction is due to spasm. The kind of operation suited to the case in hand is important, and, although it is not possible to formulate precise rules for every contingency, the following aphorisms may be of value: 1. A simple tenotomy of any one adductor or abductor muscle alone is rarely useful and seldom required; as a rule, rest under a cycloplegic combined with a full correction of refractive errors will relieve the *spasm* of a single muscle, and so avoid the necessity for a solitary tenotomy. 2. Tenotomy of a single sursumductor alone is frequently of value. 3. Advancement or shortening of a tendon, with or without tenotomy of the chief opposing muscle, should be done in most cases of abnormal deviation in the horizontal plane. 4. Where advancement (or tendon-shortening) on one side is insufficient to correct the error the same operation on the other eye is preferable to tenotomy. 5. When possible, operations should be done under a local anæsthetic, and that method chosen in which provision is made for increasing or diminishing the operative effect both during and after the operation. 6. The probable effect of the operation should be tested (red glass, cover test) during its progress. 7. Whether an overcorrection or a partial correction of the deflection is preferable will largely depend upon the refractive condition and the occupation of the patient. 8. The more the capsular attachments, check ligaments, and muscular fibres are disturbed or included in the tenotomy or advancement, the greater will be the effect upon the rotation of the globe.

Duane lays down the following rules, that differ in some respects from the foregoing: (*a*) In convergent squint due to overaction of one or both interni, tenotomy of one or both interni; when due to weak externi, advancement of one or both interni, with tenotomy of the latter; (*b*) in exophoria due to overaction of one or both interni, tenotomy of the externi; when due to insufficiency or paresis of one or both interni, advancement of the latter, combined, if necessary, with tenotomy of the externi; (*c*) in non-comitant hyperphoria (where the angle of the two visual lines constantly varies) due to weakness of the superior or inferior rectus, advancement of the weak muscle; when due to overaction of the superior or inferior rectus, tenotomy of the overacting muscle; when due to insufficiency or paresis of the superior oblique, tenotomy of the inferior rectus of the other eye; when due to overaction of the superior oblique, advancement of the inferior rectus of the other eye; when due to weakness of the inferior oblique, tenotomy of the superior rectus of the other eye; and when due to overaction of the inferior oblique, advancement of the superior rectus of the other eye. When the deflection of the

non-fixing eye has constant relation to the fixing eye (comitant hyperphoria), the best remedy is generally tenotomy of the superior rectus of the higher eye.[1]

Every operation on the eye muscles should be done under aseptic conditions. A 2 per cent. holocaine solution or cocaine (4 per cent.) is the ideal local anæsthetic, while adrenalin (1:1000) or some other suprarenal capsule preparation will give an almost bloodless field of operation. It is as yet undecided whether these agents favor a post-operative hemorrhage that may interfere with the success of the operation.

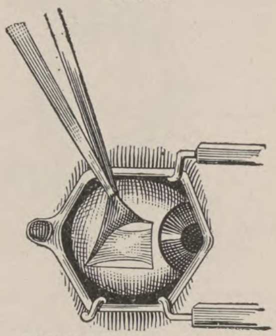
Tenotomy. A speculum (or two lid retractors held by the assistant) is inserted and a fold of conjunctiva and capsule immediately over the central insertion of the muscle *firmly* grasped by fixation forceps having at least four teeth. The underlying structures are now drawn slightly away from the globe and an incision is made with the tenotomy scissors, care being observed not to cut through the tendon itself. Sufficient space should be given to enable the surgeon to pass a strabismus hook above or below the exposed tendon, so that its point presents at the opposite border. A snip of the scissors, one blade of which is also passed beneath the muscle, now severs the tendon as near its insertion as possible. If he prefers it, the operator may proceed as for partial tenotomy (making a "button-hole" or entirely central opening in the tendon) and complete the central incision toward each margin. If, on testing, the first result is deemed insufficient, the wound in the capsule and conjunctiva is enlarged and the supplementary fibres on both sides of the tendon are carefully and gradually divided on the hook, several deviation tests being meantime made. As a rule, 5 to 10 prism degrees of deviation (or less) are obtained by a simple tenotomy where the retaining lateral fibres are undisturbed. If the capsular attachments and check ligaments are undermined and divided, a greater (and unknown) effect follows even to marked limitation of the excursions produced by the muscle operated on. It is not necessary to suture the wound. The after-treatment consists of cold applications every two or three hours, followed by a simple collyrium, such as two grammes each of boric acid and borax in 100 grammes of a 1:10,000 solution of mercuric chloride. If, not later than forty-eight hours after the operation, an overcorrection be found, a suture including the cut end of the muscle, Tenon's capsule, and the conjunctiva, should be so placed that the over-defect is remedied. If excessive bleeding occur, it is better to postpone the operation, chiefly because it is then difficult to estimate the final effect of the tenotomy. Bandages are objectionable since they prevent the use of the eyes in binocular fixation—an exercise that should begin immediately after the operation.

Advancement. The tendinous insertion may be brought forward with or without resection of a portion of the tendon itself, or the latter

[1] American Text-book of Diseases of the Eye, 1899, pp. 521–522.

may be shortened by making in it a "tuck" or "knuckle." Of the numerous operations for simple advancement, the writer has for many years been satisfied with a modification of the well-known operation of Schweigger, combined with the Black method of tying the sutures. It is usually done under a general anæsthetic. A full curved needle is threaded with No. 3 iron-dyed silk, bringing the ends of thread together and tying them in a small hard knot, or both ends of the thread may be passed through the eye of the needle at the same time, leaving the end of the suture in the form of a loop, instead of a knot. The needle is now passed through the conjunctiva, taking a good bite into the sclera close to the cornea, as indicated in Fig. 93.

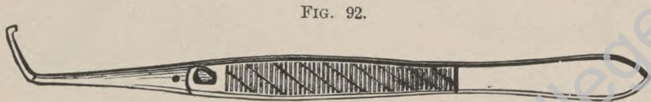
Fig. 91.



Advancement of a muscle. Exposure of the muscle. (Hansell and Reber.)

After the thread is pulled about half-way, the needle is passed through between the threads on the other side of its entrance into the sclera, and then drawn home, thus affording a firm point of fixation. A similar suture is fixed in the same manner upon the opposite side of the cornea. The conjunctiva and Tenon's capsule are now well divided over the muscle, the latter being thoroughly exposed and well cleaned of connective tissue. Two strabismus hooks are passed underneath the muscle (one from each side), or an advancement forceps (Prince's or Clark's) is made to grasp the muscular body, so as to hold it steady and away from its bed. The sutures are now passed through the muscle from below upward as far back

Fig. 92.



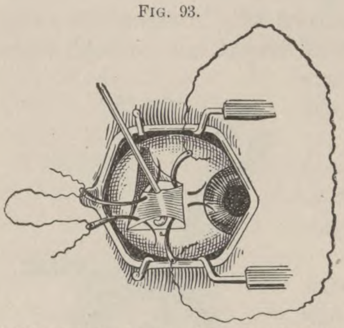
Prince's advancement forceps.

as is believed necessary, and pulled about half-way home. The muscle, still held with the hook or forceps, is now cut off just in front of the entrance of the sutures. The piece of tendon attached to the globe is grasped and cleanly dissected out.

The sutures are now pulled home, and both grasped between thumb and finger, while the globe is fixed with forceps on the nasal side of the cornea and turned outward (in operating on the external rectus), while the muscle is advanced to the desired position. The stitches are now tied in a surgeon's knot over the muscle, as indicated in Fig. 94. The original opening in the mucous membrane is stitched together by fine sutures. There may be some reaction following this operation, requiring the frequent application of hot fomentations, but if proper precautions have been taken this is unusual.

One of the most effective methods of *shortening the muscle*, by taking a "tuck" in its tendon, is comprised in an advancement operation devised in part by Frank C. Todd. Supposing the internal rectus to be operated on, a flap of conjunctiva and Tenon's capsule is dissected up and turned back, so as to freely expose the tendon. (Figs. 96 and 97.) The upper and crossed prong of the "tucker" is inserted beneath the tendon and the arms of the instrument separated by the screw-nut to produce the desired effect, as shown in Fig. 95. Catgut sutures are passed above and below, through the three layers of tendon and tied, as in Fig. 98; two double-threaded black-silk sutures are passed (one above and one below) through the loop in the tendon, thence through the conjunctival flaps and episcleral tissue on either side of the cornea to close the wound, and act as guy-ropes while the healing process goes on.

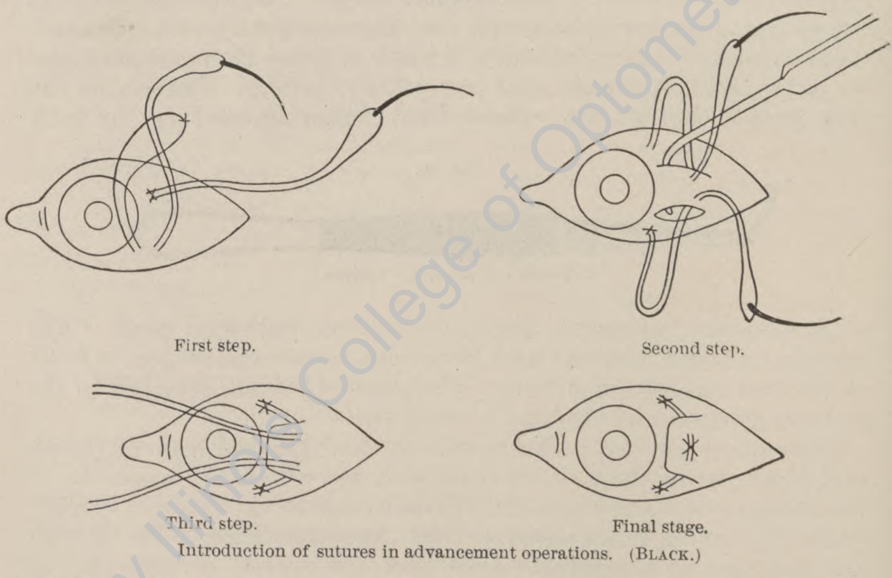
FIG. 93.



Advancement of a muscle. Introduction of sutures. (HANSELL and REBER.)

Nystagmus. This symptom consists of involuntary movements of the globe, either rotary, from side to side (horizontal nystagmus), up and down (vertical), or a combination of these excursions. The second is the commonest variety, and it is often found in those born with defective sight—*e. g.*, in albinos, in coloboma of the choroid, and in other developmental anomalies of the visual apparatus. These patients

FIG. 94.



Introduction of sutures in advancement operations. (BLACK.)

are unconscious of the oscillation of the globe, and do not complain of it as such. That this peculiar condition may be hereditary is well shown by many observers. The writer[1] reported a family of twenty-three children and grandchildren descended from a pronounced blonde male ancestor, with perfectly healthy eyes and nervous system, and a decided brunette with myopia and congenital nystagmus. Two descendants only were brunettes; they were the subjects of marked congenital nystagmus, while the other twenty-one had healthy eyes. From these and other considerations it seems justifiable to assume that there is some fault of the co-ordinating centres in most of the

FIG. 95.

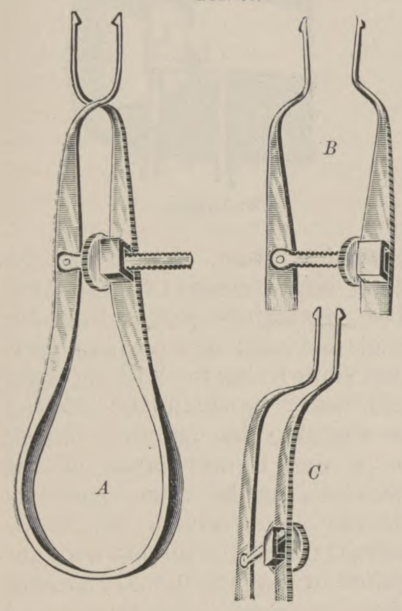


FIG. 96.

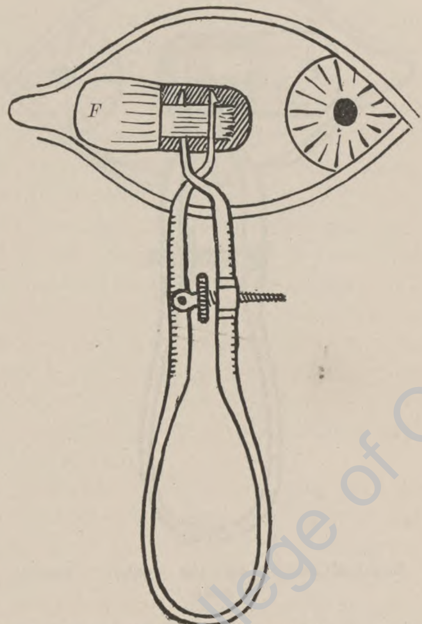


FIG. 95.—Instrument for placing a "tuck," in muscle-shortening. (TODD.)
FIG. 96.—Muscle-shortening with the Todd "tucker." First stage.

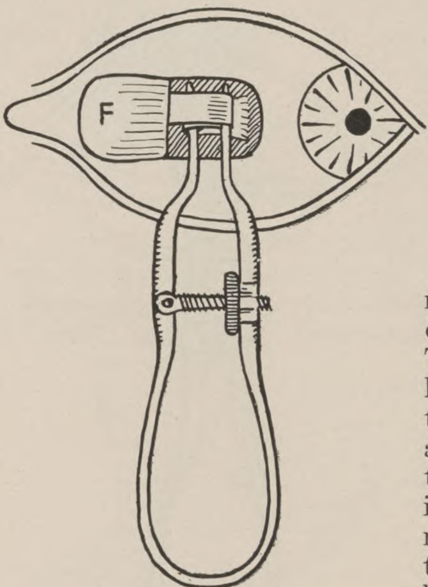
congenital cases. While visual defects are frequently present, yet not everyone with congenital visual anomalies has nystagmus, and, in some instances, the eyes, apart from the irregular globar excursions, are practically normal.

Acquired nystagmus is present in 50 per cent. of all cases of sclerosis in patches, and it is also noticed occasionally in those who work in abnormal or strained attitudes. A well-known example of the acquired variety is "miner's nystagmus." Here, to the unnatural positions these workers are obliged to assume for hours at a time, is added insufficient light; the ocular centres of co-ordination are not

[1] North American Practitioner, April, 1892.

properly stimulated, owing to a lack of definite retinal images, and the unwonted strain on the oblique muscles in particular ends in irregular movements of the globe. We thus have to deal with a professional inco-ordination of a class akin to writer's cramp, the individual so affected being both conscious of and annoyed by the trouble. Later on, his nervous system adapts itself to the situation, just as in the case of congenital nystagmics. The treatment of the congenital variety is to improve the vision, if possible, and to correct

FIG. 97.



Muscle-shortening with the "tucker." Second stage.

FIG. 98.

Third stage.

muscular errors. If this can be done, improvement often follows. The nystagmic patient usually holds his head in a peculiar position while fixing for both distance and near; he should be allowed to continue this practice (unless it be due to imbalance of his muscles), as he often succeeds thereby in steadying the oscillating eyeballs. Patients with acquired nystagmus should abandon their injurious occupation, and give their eyes prolonged rest, correcting-glasses being ordered for distant fixation. A cure generally follows if hygienic measures are early applied.

Exercise of the Weak Eye and the Use of the Stereoscope in Heterotropia. Still less than formerly is the ophthalmic surgeon content with merely "straightening" the crossed eyes of his patients. As we have seen, loss of binocular vision is involved in all cases of squint and in some classes of heterophoria. We have, consequently, not done our full duty until we have made every effort to restore or to enable the patient to acquire the capacity for seeing with both eyes together.

In quite a few examples of strabismus this desirable result is necessarily impossible. The squinting eye may be congenitally defective to a degree incapable of vision with the fellow eye, or there may be an unconquerable aversion to binocular sight upon the part of one

or both eyes, the nature of which we do not know, except that it probably predicates a lack of development in some one or other of the central neurons implicated in the visual act. Again, binocular single vision may be unobtainable on account of incurable lesions (corneal nebulæ, intra-ocular disease) of the strabismic eye, whereby sight is permanently lowered, although it may be good in the fixing eye.

Fortunately it happens that in a majority of instances binocular vision is to a greater or less extent possible after the relief of strabismus. Moreover, the eyes that acquire stereoscopic or single vision are usually those whose excursions in all directions are normal and remain normal. The ideal result, then, from the surgeon's standpoint differs from that of the patient in that, while the latter is alone interested in the cosmetic appearance, the former is concerned in the question as to whether the hitherto useless eye can be made to take part in the function of sight. The old device of covering the better eye, so that the weak (or squinting) organ may be exercised and strengthened, is helpful before operation, or as adjunct to other treatment, if carried out regularly, say, for half an hour at a time, three or four times daily. With a little patience, children of tender years can be induced to wear a light bandage over the amblyopic eye, and use it, even while at play, to great advantage. As it is highly desirable to begin the treatment of infantile strabismus at as early a period as possible this plan should be carried out, in conjunction with the periodic use of atropine and tinted glasses as soon as the child begins to walk. A single drop of a $\frac{1}{2}$ per cent. solution of atropine instilled into each eye three times daily for the first few days of each month certainly helps to relieve the early spasm of the interni and cil ary muscles and to check a convergent squint.

In quite another fashion do we endeavor to force the two eyes to functionate as far as possible in unison after the employment of means (glasses, atropine, operation, etc.) to correct the strabismus proper. The best means is the employment of the stereoscope. Landolt, Worth, and others have invented various improved instruments, and while these are of advantage, especially in private practice, none is absolutely necessary. The ordinary stereoscope is provided with a card on which are drawn or printed pictures—two such dissimilar objects as a square and a circle will do very well—one opposite each eyehole. The patient exercises for a few seconds, first of all, the defective eye alone, and then, uncovering the sound eye, looks at both objects through the instrument, endeavoring to see them singly if possible. At first it is well, as Landolt suggests, to weaken by means of lenses the vision of the better eye, and so further encourage the defective organ. Such exercises should be employed immediately after operation, and ought to be continued several times daily for weeks or months. They should also be supplemented by the exercise of single vision in the distance. A red glass is placed before the better eye while the patient attempts to fuse the red and white images of a candle placed 6 m. away.

CHAPTER V.

DISEASES OF THE ORBIT, LACRYMAL APPARATUS, AND LIDS.

BY R. A. REEVE, M.D.

THE ORBIT.

Anatomy. The human orbit consists of two cone-shaped cavities, about one and three-quarters inches deep, which slightly converge toward one another, as may be seen by the accompanying figure. (Fig. 99.) Each orbit is composed of a number of bones—*i. e.*, the frontal, the superior maxillary, the malar, the palate, the lacrymal, the sphenoid, and the ethmoid.

By reason of the organ which it contains, as well as the close connection which it has by means of its thin walls and its foramina with the cranium, the ethmoidal, the sphenoidal and frontal sinuses, and the antrum of Highmore, the orbit may well be regarded as one of the most important cavities of the body.

The posterior portion or apex of the orbit contains three important apertures: the optic foramen, for the transmission of the optic nerve and the ophthalmic artery, the superior orbital fissure, through which pass the nerves which supply the muscles of the eye, and the first branch of the trigeminus, and the inferior orbital fissure. The second branch of the trigeminus passes through this opening.

The supra-orbital notch is found at the upper inner angle for the transmission of the supra-orbital artery and nerve, while a canal just below the inferior rim of the orbit contains the artery and nerve of the same name.

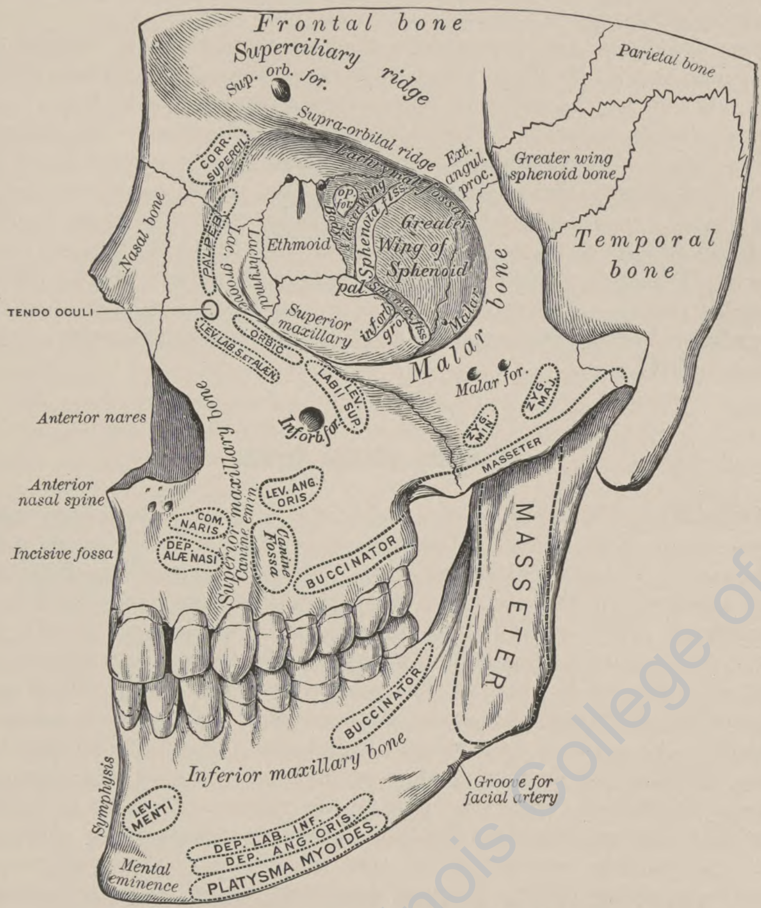
The orbit is relatively large in children, Merkel being authority for the statement that at five years of age the base of the orbit lacks only 2 mm. or 3 mm. of its adult height, which it gains usually in the next two years. Its full breadth is not attained, however, until somewhat later. The periosteum covers the walls of the orbit, and the fissures are closed in by membrane. The orbit contains the eyeball and its attached muscles, the optic nerve, the vessels and nerves, and the lacrymal gland. In addition to these structures there is a cushion of fat which fills in the interstices between them, and a dense fascia which connects all the parts and is expanded along the walls of the orbit, upon the ocular muscles, and, finally, upon the eyeball itself.

For the protection of the eye against injury, the orbit is bounded superiorly by the eyebrows, externally and inferiorly by a strong rim,

and internally by the nose. The eyebrows vary in texture, direction, and size in different individuals, but as a rule they are coarser and more marked in men than in women.

The fissure which is made by the opening of the lids, the so-called *palpebral fissure,* is oval, averaging from 25 to 30 mm. in length, and from 12 to 14 mm. in breadth. If the eyeball be protruded,

FIG. 99.



Antero-lateral region of the skull. (CRYER.)

exophthalmos, the fissure widens; if it recedes, *enophthalmos,* the fissure is narrowed.

Exophthalmos results from an increase in the volume of the contents of the orbit, as in hemorrhage, cellulitis, or neoplasms of the orbit; it may be occasioned by a lack of tone in the recti muscles, or after paralysis, or it may be a symptom of Graves' disease or exophthalmic goitre. (See page 208.)

Enophthalmos. Recession of the eyeball occurs after injuries in which cicatricial contraction of the orbital tissues has followed, or atrophy ensued, from nerve lesions, and in cases of fracture with displacement of the floor of the orbit (Lang); when the orbital contents have been reduced in the course of operations or in cholera (from excessive drainage), and in marasmus; also in paralysis of the sympathetic involving Müller's lid-muscle; after the spontaneous subsidence of pulsating exophthalmos (Bronner), and in the neurotic atrophy of the face.

Enophthalmos also occurs in a class of cases recently worked out by Tuerk and by Wolff, in which there are congenital shortness, fixity, and inaction of the external rectus of one or other side, from "paralysis" (really aplasia). The normal tonic contraction of the internal rectus draws the eye slightly backward, and causes narrowing of the palpebral fissure, owing to loss by the lids of the usual support of the globe. In attempted adduction the retraction is more marked and the fissure narrower, and the globe is apt to be turned up or down. Treacher Collins says the tonic contraction of the orbital muscles (which are congenitally short) is not compensated, owing to the absence or insertion too far back of the check ligaments; hence the enophthalmos.

Diseases of the Orbit.

Cellulitis. This may be of a mild type, and soon abate, but much more often it is severe or phlegmonous, and ends in suppuration (abscess). The early symptoms are redness and swelling of the lids and chemosis of the conjunctiva, with deep-seated pain and some tenderness. The mobility of the eye may be impaired. At this point resolution may begin (first group), or the disease may be arrested and recovery set in; but in the usual course the inflammation goes on steadily and rapidly, the temperature rises, and the pain becomes intense. As early as the third or fourth day the greatly swollen lids may be distended or even pushed apart by the highly engorged and chemotic eyeball, which is now quite prominent and immobile. The lids become brawny, and soon there is indication of pointing, and spontaneous evacuation of pus may occur. The vision may remain good, or it may become defective, owing to optic neuritis, etc. The degree of stretching of the optic nerve from *proptosis* (forward displacement), compatible with normal vision, is often a matter of surprise.

Etiology. Cellulitis may be caused by exposure, cold, etc.; trauma, blows, penetrating wounds, especially with septic invasion, lodgement of foreign bodies; lacrymal cystitis, erysipelas, anthrax, etc.; septic emboli of the orbital veins, as in pyæmia, metria, etc.; periostitis and osteitis of one or other wall, as in sinusitis of the ethmoid, frontal, or antrum, and suppurative periodontitis; metastasis, in which the

pneumococcus is the active agent; also septic phlebitis and thrombus in thrombosis of the cavernous sinus. There are at times points which aid in the diagnosis, *e. g.*, if in a case of purulent rhinitis (influenza, la grippe), with ensuing œdema, mainly of the inner end of the lid, chemosis and impaired adduction, acute cellulitis should develop, it might fairly be ascribed to ethmoiditis, which would cause an exudation on the inner orbital wall that would cripple the internal rectus. So if the lower lid became first affected, the eye being pushed up and rotation down restricted, disease of the antrum would be suspected. If the cellulitis is symmetrical (double), or that of the second eye follows after a short interval, a diagnosis of thrombosis of the cavernous sinuses, likely septic, would be warranted.

Treatment. If the case is seen early, the ice-bag or iced compresses should be ordered, with local depletion by leeches or wet-cups at temple; if the leeches are applied at the inner canthus, the bleeding is more effective. The patient should be put to bed; small doses of calomel with anodynes and salines may be given, and any faulty condition of the nasal passages attended to. If relief is not had, the hot fomentations should be substituted. And if there is reason to suspect the formation of pus, or the eye itself is suffering, deep incisions should be made, preferably through the conjunctiva, but if the lid cannot be pressed back, then directly through the lid. Great care must be taken to avoid the globe, the point of the long, narrow scalpel or linear cataract knife being pushed slowly in and deeply along one or other bony wall of the orbit. The adult orbit is one and three-quarters inches in depth, and, as pus sometimes forms near the apex, the knife should go deeply if needful. If pus does not escape, a second or even third exploratory puncture should be made at other points. The wounds should be kept open by tents, and if pus presents, syringing should be practised daily with 1: 3000 solution of perchloride, or 1: 40 carbolic acid, etc. Where empyema of the ethmoid is found, it is desirable, in some cases at least, to open a passage by means of a strong probe or trocar into the nasal fossa through the ethmoid, drawing the drainage-tube through by means of a thread attached to the eye of a probe. In this way more effective irrigation of the parts can be made. Necrosed or carious bone will require curetting, and any disease of the antrum, frontal sinus, lacrymal sac, etc., should be treated.

Periostitis and Osteitis. These may be acute or chronic. The acute form may be caused by extension of inflammation from adjacent parts, most often the ethmoidal or frontal sinuses, the signs indicating the probable point of origin; also by blows, foreign bodies, etc., the point of impact determining the site, generally in the region of the orbital border. Slight injuries may be effective in young scrofulous subjects, in whom osteoperiostitis may also appear to be idiopathic. Syphilis may set up an acute localized periostitis, and cellulitis may also cause it. Acute periostitis and osteitis may end in resolution, but more often suppuration (abscess) develops, or they

become chronic. Chronic periostitis and osteitis are, as a rule, due to syphilis (tertiary). The orbital rim is the most common seat of the at first ill-defined doughy tumors (nodes) attached to the bone, which are attended by characteristic nocturnal pain or increase of pain. The infiltration and pain soon yield to large doses of potassium iodide. Left alone, ulceration and caries may set in, causing sinuses in the fascia and lids, with final deformity (ectropion, entropion, etc.). Nodes may also undergo eburnation, either from chronic periostitis (periostosis) or condensing osteitis with hypertrophy (exostosis). A deep-seated periostitis now and then occurs, mostly syphilitic and tertiary, which causes paralysis of the orbital muscles by involving the third, fourth, or sixth nerve, and it may also cause exophthalmos, partly hidden by the ptosis. When at the apex the optic nerve may also be involved, the pressure or neuritis causing amblyopia. Periostosis may ensue, and in this event, or if treatment be too late or ineffective, there will likely be permanent blindness from secondary atrophy of the optic nerve, with ptosis and other paralysis. Very rarely periostitis at the apex with some cellulitis and pure proptosis are due to empyema, etc., of the sphenoidal sinus. Secondary amblyopia and contracted field, or optic neuritis, atrophy, and blindness are apt to occur, and may be double. Pain in frontal, temporal, and occipital regions, variable vision, and the results of rhinoscopy and ophthalmoscopy, may point to the diagnosis. In periostitis pressure on some part of the orbital rim causes much pain, and the œdema of the lids develops less regularly than in cellulitis proper. In the former, signs of the disease are much more apt to be localized, *e. g.*, one lid or even part of a lid may be swollen, etc., and the bone only tender beneath it. (Fig. 100.) In cellulitis pressure on the globe itself is apt to cause deep pain, and palpation just within the bony rim shows that the parts are firm, tense, and tender. Very rarely acute *diffuse* periostitis occurs, and cellulitis quickly ensues. The systemic disturbance is greater than in acute cellulitis, and the condition is much more grave. As suppuration is apt to occur rapidly, with added risk of necrosis and burrowing sinuses if abortive treatment fail, early incision to the bone is indicated, and several will be needed in the diffuse form.

FIG. 100.

Periostitis of orbital margin.

Periostitis of one or other wall of the orbit which stops short of exciting acute diffuse cellulitis is not uncommon. In chronic, quiet ethmoiditis with purulent discharge from the nares, osteitis of the os planum, and secondary periostitis by extension on the orbital side, the cellulitis and exudation may be limited, the œdema being mainly in the nasal half of the upper lid, with impaired adduction and probably slight displacement of the eyeball outward, with diplopia. In the same way periostitis of the upper orbital border may follow

osteitis of the floor of the frontal sinus in chronic or acute sinusitis, with secondary œdema of only the upper lid, mainly at the inner half. Under these conditions pressure on the bony rim is painful, and the eye may be displaced slightly down and out. If the antrum is the seat of the primary disease, the lower margin of the orbit will be very tender, and œdema mostly of the lower lid will develop, movement of the eye downward being restricted, or the globe may be tilted upward.

Treatment. In adults local depletion by leeches or wet-cups, the ice-bag, the Leiter coil, or compresses wet with the lead-and-spirit lotion, rest in bed; in rheumatic cases sodium or strontium salicylate in frequent doses with or followed by potassium or sodium iodide, and in these subjects dry heat by Japanese hot-box or hot fomentations may be more comforting than cold; in secondary cases a short active course of mercury by inunction or internally, with potass. iodid. in ascending doses, and in those at the tertiary stage pot. or sod. iodid. in doses of fifteen grains, thirty grains, to sixty grains every four hours. If signs of pus show, early incision to the bone is indicated, followed by a course of gentle syringing with antiseptics, *e. g.*, carbolic acid 1 : 100 or 1 : 40, hydrarg. perchlorid. 1 : 3000, etc. Syringing should be kept up as long as there is any purulent discharge, and a tent or tube used until it is evident the disease is spent. In so-called scrofulous cases syr. ferri iodid. and syr. calcis lacto-phosph., syr. phosph. (Parrish), ol. morrhuæ, with the best hygiene, are indicated. In case of osteoperiostitis where there has been spontaneous opening the probe should be used carefully so as not to disturb unduly nature's barriers and cause further complication. All rough bone is not necessarily necrotic, and time should be given for necrosed tissue to separate and the carious surface to heal over before one actively interferes. Then, if needful, the sinus should be enlarged by sponge tent or incision, and the curette, gouge, etc., used *secundem artem*.

Tenonitis. This is, as a rule, a mild type of inflammation of the capsular portion of the orbital fascia. The symptoms are chemosis and exophthalmos, which may be slight; pain and tenderness on pressure, with impaired mobility. Œdema of the lids is absent in slight cases, and is not a marked feature of any. This is one point in diagnosis between tenonitis and cellulitis proper—in the latter the œdema of the lids is marked. The chemosis of tenonitis is also distinct, and at times decided, and is always out of proportion to the œdema. The reverse is the case in cellulitis. It is due to rheumatism and gout, mild sepsis, and erysipelas, and always follows panophthalmitis, which may also excite cellulitis; it is sometimes caused by injury and, now and then, follows tenotomy.

Treatment. In the tenonitis of rheumatic or gouty origin marked and speedy relief follows the prompt and full exhibition of sodium (or strontium) salicylate with or without colchicum; potassium or sodium iodide, and lithia being given at the same time or later. Pilocarpine

or jaborandi in dose sufficient to cause free sweating, daily for a few times, may suffice alone, and it may be used as an adjuvant to hasten recovery. Topically, the lead-and-spirit lotion may be useful, and, where dry heat is preferred, the Japanese "hot-box."

Injuries, Foreign Bodies, etc. Foreign bodies most often enter between the globe and the roof or the inner wall. A large foreign body may enter and be impacted and the eye escape. Hence an off-hand opinion should not be given, especially in view of the tolerance of the socket for intruding non-septic bodies. Many notable examples of the latter could be cited, curious, interesting, and instructive.[1] On the other hand, the possible risk to life from secondary processes in seeming slight injuries should be kept in mind, and therefore, as a rule, a guarded prognosis be given, especially in view of a possible medico-legal bearing. The *primary* effects may include injury or loss of the eye, or lesion of the optic nerve, with sudden blindness, or true aneurism, or aneurismal varix, or free hemorrhage with infiltration of the tissues, proptosis, etc. The orbital wall may be penetrated or fractured. Following forcible entry of a pointed stick, foil, etc., especially from below, perforation of the roof with lesion of the meninges or brain is apt to occur. Bullets may enter through the temple and cut the optic nerve, or plough their way through globe or lids and on into the anterior or middle fossa. Severe blows may fracture one or other wall of the orbit or of the optic canal, cause hemorrhage into the sheath or direct lesion of optic nerve and blindness, without external sign, or bleeding into the cellular tissues, with extravasation into the lids, when fracture is almost certain; or may open communication with one or other sinus, causing emphysema, with much swelling and crackling crepitus. Some large foreign bodies lodge in part in adjacent cavities, as the sphenomaxillary fissure, nasal fossa, etc.

The *secondary* results of injuries are orbital cellulitis (generally septic), with possible optic neuritis and atrophy, and blindness; paralysis of one or more ocular muscles, periostitis, and osteitis, with consecutive meningitis, brain abscess, and death; also thrombosis of the cavernous sinus, single or symmetrical, from the septic orbital phlebitis of cellulitis; and enophthalmos. It should be borne in mind that penetrating wounds with lesion of the roof are treacherous; a quiet interval of several days or even weeks with fair promise may end abruptly in grave acute symptoms with speedy death.

One may get some clue to the lesion from the nature of the accident and the symptoms, and by the careful use of the probe and finger. Great pains should be taken and will be rewarded, while care is had not to add to the trauma. Where an interval has elapsed, a spongy cicatrix, a wound which will not quite heal, or one which reopens, is significant. At times several foreign bodies may be lodged. Some

[1] Of recent ones: knife-blade, 38 mm. long by 8 mm., in lower part of orbit thirty-two years, with normal vision and perfect movement of the globe. (C. Holmes.)

cases are not obscure, *e. g.*, an impacted splinter in the outer wall may cripple the external rectus, causing pain in attempted abduction, with convergent squint and diplopia. A skiagraph after the improved methods of Sweet, Davidson, and others is, of course, decisive as to the site of metallic missiles or other foreign bodies opaque to the x-rays.

Treatment. Small bodies unless easily got at are best left *in situ* if not causing trouble. Large bodies should be removed promptly, noting on a trial attempt if the globe is dragged upon. Some freeing or careful dissection may be necessary, the wound being enlarged; and in case of impacted arrow-shafts or a large splinter, strong forceps are required in lieu of the sequestrum or crocodile pattern, which usually suffices. Iced compresses, cold lead-and-spirit lotion, rest in bed, salines, etc., will tend to ward off undue reaction. If pain and swelling persist or light up, with pyrexia, in spite of these and local depletions, and pus seems forming, hot fomentations should be used, and incision made as in cellulitis and periostitis.

Pulsating Exophthalmos. This is a condition largely due to trauma, in which proptosis and pulsation of the eyeball and loud subjective and audible tinnitus are leading symptoms. The old-time view as to the etiology is not now held, for there can be no doubt that it is most often due to intracranial and not orbital lesions, the changes in the orbital contents being secondary and symptomatic. In the few cases in which the mischief has been proved to be orbital there have been found true aneurism of the ophthalmic artery or of some of its branches, traumatic diffuse or circumscribed aneurism, and arteriovenous aneurism, aneurism by anastomosis and angiomata, or telangiectatic tumor. Of intracranial lesions, the most common is traumatic aneurismal varix in the cavernous sinus, the carotid artery pumping into the sinus through a breach in its wall, and so into the ophthalmic vein, etc.; aneurism of the internal carotid, aneurism of the ophthalmic artery at its point of origin from the internal carotid—very rare.

Fig. 101.

Traumatic pulsating exophthalmos.

Symptoms. SUBJECTIVE. One-sided loud, pulsating, blowing, churning, rushing, or roaring tinnitus, increased by stooping, reduced by opening mouth widely, and stopped by compression of the common carotid of the same side; diplopia often, at least in some parts of the field (*e. g.*, paresis of external rectus); pain not a constant symptom, but present in many cases.

OBJECTIVE. Proptosis, sometimes very marked, with inability to close the eye, and at times displacement outward and downward, with impaired motility; convergent squint constant or on attempted abduction; eyelids dusky and much swollen, and veins of brow, fore-

head and temple notably distended and quite sinuous; a tense but compressible vascular tumor at inner end of brow—dilated ophthalmic vein—giving a strong impulse to finger-tip; vessels of eyeball much engorged and conjunctiva chemotic; caruncle large and fleshy and protruding. (Fig. 101.) Palpation of lids and globe yields a thrill and distinct pulsation, the latter visible. There is a loud bruit with ear to side of head or at any point of it, loudest with stethoscope at inner end of brow, with instant almost startling quiet on compression of common carotid. The latter stops all thrill and pulsation, the eyeball readily yields to pressure, and the finger may be dipped deeply into the flaccid ophthalmic vein. The sight and field of vision may be normal or but little affected. The fundus shows hyperæmia of the optic disk and pulsation of the retinal veins, which are apt to be greatly dilated and tortuous. There are at times papillitis and retinitis, etc.

Etiology. Pulsating exophthalmos may be idiopathic or traumatic. The former occurs mainly in females, the latter most often among men; while in some of the traumatic cases the symptoms appear quickly, in others they develop slowly. In the idiopathic variety they are apt to be sudden in onset—pain and a great "crack" or "snap" at the start, and in a few hours great swelling of lids, œdema of conjunctiva, proptosis, and pulsation, with most disturbing tinnitus. When due to trauma, an early if not the first special symptom in some cases is a high note as if there were only a small hole in the vessel wall. The loud, pulsating, and roaring tinnitus may not ensue for some days or weeks, and the same interval may mark the other main symptoms. Aneurismal varix in the neck may cause pulsating exophthalmos by damming the venous outflow from the sinuses. (Gifford.) The diagnosis should be much aided in the idiopathic cases by the rapidity and fulminating nature of the symptoms. The fact of trauma, more or less severe, and of the tense pulsating sac at the upper inner part of the orbit, at once made flaccid by stopping the carotid flow, should distinguish from orbital growths, especially vascular malignant forms, which also cause proptosis and pulsation. Then, paresis of the *external* rectus, with pulsating tumor on the *inner* side of the eyeball, is significant. Some points in the *pathology* have already been cited. There are some anomalies; the absence of aneurism or other vascular lesion has been shown in a series of cases by autopsy. And in various instances of true aneurism of the internal carotid in the cavernous sinus, as proved by post-mortem, pulsating exophthalmos had not occurred. Relief of pressure on the ophthalmic vein through the setting up of collateral circulation might explain this.

Treatment and Prognosis. In view of the fact that there has been spontaneous recovery in probably 7 or 8 per cent., some surgeons follow an expectant course, giving potass. iodid., and enjoining quiet, etc. Compression of the common carotid has cured in a still larger number, and rightly is held worthy of trial. It is resorted to by some merely as a safeguard before operating. If kept up for

a short time every day for weeks or months in idiopathic cases, it may succeed; in traumatic cases it should be applied continuously for hours daily. (Sattler, in Noyes.) In a case of the writer's, of single pulsating exophthalmos due to trauma, in which ligation of both common carotids proved ineffective, it was preferred to test first the effect of pressure in conjunction with a course of potass. iodid., because there were no urgent symptoms present; the sight was good, there was no pain, the process was seemingly at a stand-still, and compression had availed in various cases; while, on the other hand, ligation had sometimes failed, and was itself not free from risk.[1] Ligation of the common carotid is the most reliable treatment, being effective in about 60 per cent. of the cases operated on. Death has followed in about 10 per cent. of the remainder.

Ligation of the second common carotid has now and then been curative in failure of ligation on the affected side. Ligation of the external carotid on the affected side after failure of that of the common carotid has sometimes been successful, and in the writer's opinion it is a step that should be taken in preference to ligation of the second common carotid. There seems ground for the view that treatment by prolonged compression militates against the success of ligation. As a rule, life is not jeopardized, although rendered miserable in cases which have been left alone. The attempt to plug the ophthalmic vein by a firm, deeply placed clot by means of galvanopuncture, the positive pole (needle) being passed far in, is worthy of trial, and it should prove useful, if not *per se*, at least with compression, medication by potassium iodide and ferric tannate, or where ligation has given only partial relief.

Dr. Argyll Robertson reports[2] two cases of pulsating tumor of the orbit with bruit, in which electrolysis was used.

Thrombosis of the cavernous sinus causes venous stasis in the orbit, with general infiltration and œdema of lids. If due to sepsis, as is the rule, phlebitis and thrombi with cellulitis follow, with marked œdema of lids, exophthalmos, and immobility of globe. Before the eyeball becomes fixed, paralysis of the third and sixth nerves may be made out. The pupil is generally dilated. There may be great tortuosity or thrombosis of the intra-ocular veins, with impaired sight or blindness.

Etiology. Thrombosis of the cavernous sinus is generally septic in origin and due to purulent or carious foci, likely infective, in some part of the head or neck; most often a sequel of infective inflammation of the lateral sinus, with septic thrombosis from suppurative otitis, with or without caries of the petrous. Erysipelas may set up thrombosis through the medium of orbital phlebitis or cellulitis; also septic foci may cause it in the nasal fossa, lacrymal sac, or cheek, as in anthrax or malignant pustule. Thrombosis of the second cavernous

[1] Transactions of Ophthalmological Society, 1893.
[2] Transactions of the Ophthalmological Society of the United Kingdom, vol. xx.

sinus occurs by extension from the first through the circular sinus, or directly, as in erysipelas. This contingency adds a feature of gravity to facial erysipelas which should be borne in mind. In thrombosis there would be the grave general condition, weak, quick pulse, temperature showing steep-peaked chart, with probable rigors and hebetude, the history, likely, of chronic otorrhœa or recurrent otitis, with œdema and tenderness over and behind the posterior edge of the mastoid, and double optic neuritis. To make a correct diagnosis is to give a bad prognosis and to avoid the error of treating the condition as cellulitis *per se*, for septic cases are, as a rule, speedily fatal, and when double, death is the more certain. In the early stage antitoxin treatment, or the use of Crédé's ointment, collargolum, etc., may prove of value.

Tumors. The anatomy of the orbit, its constituents and relations, render it prone to become the seat of neoplasms and to be invaded by growths of adjacent parts. Any change in the bulk of its contents or its capacity is apt to alter the position of the eyeball, which is a sort of movable plug in the septum orbitæ. Hence prominence or

FIG. 102.

FIG. 103.

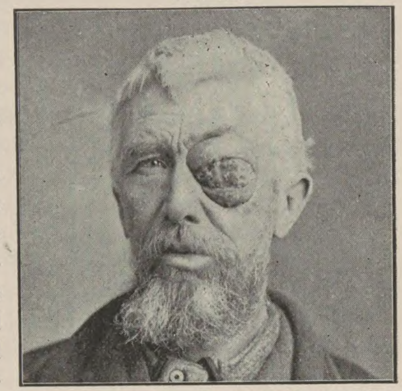
FIG. 102.—Sarcoma of orbit. Twenty-one inches in circumference. Weight, three pounds.
FIG. 103.—Ivory exostosis of F. Sinus and orbit with marked exophthalmos.

protrusion of the globe, *exophthalmos*, is the most common sign and result of orbital hyperplasia and tumor, and it often depends upon the same changes in adjacent cavities, with distention, etc. When the globe is pushed straight forward or in the line of axis, the term *proptosis* is used; displacement is generally lateral as well as forward. A displaced eye may functionate normally and give no trouble, but crippling of one or other of the orbital muscles, directly by pressure or invasion of its fibres, or through the motor nerve, at once causes disability. The ease with which paresis is induced renders it a common result of orbital lesion (as well as intracranial). In addition to exophthalmos and defective motility, œdema of the lids and at times great stretching are incidents of orbital growth. Pain, which is often wanting, may be most severe and annoying if there is much tension of the parts.

Tumors of the orbit may, as elsewhere, be benign, recurrent, or malignant. To the benign class belong fibromata, osteomata, cysts,

vascular tumors (angiomata, etc.), and lymphomata or lymphadenomata. Malignant growths, gliomata, carcinomata, sarcomata, etc., are, of course, apt to recur, the round-cell sarcoma of children practically certain; but recurrence does not stamp malignancy, for vascular and cystic neoplasms are prone to grow again unless eradicated. A fatal result is brought about by extension to the brain along the optic nerve, or through the sphenoidal fissure, or through the walls, *e. g.*, roof by caries or by metastasis. It is often delayed, and may be averted in orbital tumor from the great tendency in such cases to growth outward, *i. e.*, forward. (Figs. 102, 103, and 104.) The division may be arbitrary, but is useful, of orbital tumors into those arising within the socket and starting from the cellular tissue—the main seat, the walls, lacrymal gland, optic nerve or vessels—and those invading the orbit from adjacent parts or cavities, as is often the case in osteomata and sarcomata. In the latter in young subjects, without pain or pyrexia, there may be no sign until exophthalmos shows, and then there is rapid growth. One should weigh well such points as the condition of the patient, the size and the rate of growth, site or origin and attachments of the neoplasms, whether fixed or movable, hard or soft, smooth or nodular, compressible, tender or pulsating; the kind and degree of displacement of the globe, and of any paresis, the acuity of vision, state of the fundus oculi, and of the adnexa and accessory sinuses. As growths vary so much in nature, and if malignant should be extirpated early, if at all, it is advisable to employ every diagnostic aid. Inspection and the careful use of the finger can tell one much. The finger-tip is pressed along and just within the orbital rim, and then back between it and the globe, testing the floor of the frontal sinus, the region of the ethmoid back on the inner wall, of the lacrymal gland, and of the antrum. Cysts may feel firm when well packed; soft, round-cell sarcoma if encapsulated, may stretch the bag so tightly as to seem quite hard, and a subperiosteal mass of the same, or even of pus, may simulate hyperostosis. Hence the need of exploratory incision to determine the true state of things, especially when too deep for palpation. Under asepsis it is free from special risks, and it should always be made in cases of doubt. An incision is carefully made through the skin in line with the orbicularis fibres, and just within the orbital margin, or between the lids and the globe, going deeper and slowly, and if beyond one's ken the mirror may be used,

FIG. 104.



Sarcoma of orbit. Encephaloid enteration. Living thirteen years afterward. No recurrence.

also the little finger and probe. Exploratory puncture at times gives a useful hint, and the microscope may be needed to fix the diagnosis. Krönlein's osteoplastic resection of the outer wall of the orbit has been urged and used for diagnostic purposes. It has proved useful in exposing and removing deep-seated neoplasms with the least risk to the globe, optic nerve, etc. Eyes may require to be sacrificed by operating from the front which could be saved by the Krönlein method. This granted, good work can be done and large growths removed by incisions through the lids. It is important to learn the state of the nasal passages by anterior and posterior rhinoscopy, and of the maxillary sinus—at least by exclusion—before giving the prognosis or resorting to operations. Transillumination may be of service, for if the antrum or frontal sinus fail to light up there is likely a growth or other morbid condition.

Angioma may begin in the lids and dip into the orbit, and it may start in the orbit and invade the lids, involving also the lacrymal gland and requiring sacrifice of the latter in the extirpation (de Schweinitz). The rare *encapsuled cavernous* variety of angioma simulates tumor of the optic nerve, but vision is unaffected. It is removed by careful dissection. The Krönlein method is preferable.

Aneurism by Anastomosis. Subcutaneous nævus is a species of angioma in which congeries of dilated arteries from a mass that yie ds pulsation and a tough and doughy feel on palpation, and gives a bluish tinge to the swollen lid.

To a variety of angioma, *varicose veins* in the orbit, is attributed the rare *intermittent exophthalmos,* in which protrusion of the eyeball occurs when the head is dependent, and recession in the erect or recumbent position.

Lymphangioma is allied to cavernous angioma. It is a retro-ocular encapsuled growth, and is similarly treated by extirpation.

Pure myxoma occurs now and then as a soft or doughy encapsuled tumor, causing exophthalmos, etc., and should be dissected out.

Lymphoma or *lymphadenoma* is an occasional innocent neoplasm simulating clinically and histologically small-cell sarcoma, but yielding to a full course of arsenic. This remedy should have a fair trial in all doubtful cases as should potassium iodide whenever there is the least suspicion of specific origin.

Lipoma or encapsuled fatty tumor is of very slow growth. If small, it may be felt as a soft, elastic, movable tumor; if large, the eye and lids will be more or less prominent, and the latter yield a fleshy feel. It is very rare, and excision is the only treatment. Fibro-angiolipoma is not unusual (W. A. Holden).

Chondroma or *enchondroma,* a purely cartilaginous tumor, is exceedingly rare. It may spring from the sphenoid, and, growing very slowly, cause at length great exophthalmos. Now and then chondrosarcoma and chondrofibroma occur.

Tumor of the Optic Nerve. The features are slow and, as a rule, painless growth in a healthy subject, gradually increasing proptosis,

mostly in the line of the axis of the orbit,[1] but with mobility of the eyeball probably good, optic neuritis, or "atrophy," and vision early impaired or lost.[2] In some cases marked hypermetropia develops from flattening of the globe by retro-ocular pressure. Palpation may not make out the tumor if small. Removal is indicated. The neoplasm is encapsulated and non-recurrent, and therefore the prognosis is so far good; but it is likely a much higher percentage succumb to intracranial extension—possibly after a long interval—than is suspected. Fibromatosis expresses the pathological character of these growths, which histologically are essentially mesoblastic in nature (Byers). (See page 464.)

Operation. Extirpation may be done by careful deep dissection on the inner or outer side of the globe with the finger-tip as a guide (H. Knapp's method), the closed scissors' points being used to separate parts and to isolate the growth. The external rectus and outer canthus may with advantage be cut to gain access (Lagrange), to be reunited after removal. The optic nerve is cut close to the apex, traction is made, the neoplasm detached from the globe if in contact, the nerve then divided next the eyeball, and the growth brought away. Some prefer to section the nerve in the reverse order. The eye sometimes is saved, and remains cosmetically good, but more often it is either sacrificed or finally shrinks. The Krönlein method gives freer access to the orbital contents than the above method, long in vogue, and with less risk of excessive hemorrhage and of loss of the eyeball.

Kronlein's Method. A slightly curved incision is made along the outer bony margin of the orbit, dividing the periosteum. The periosteum lining the inner side of the lateral wall of the orbit is retracted, together with the soft orbital contents, and the inferior orbital fissure localized. From the anterior end of this fissure the bony wall of the orbit is cut through with a chisel along two diverging lines, the one passing up and out to the external angular process of the frontal bone, practically in the suture between the great wing of the sphenoid and the malar bone, and the other in a horizontal plane passing out and forward, appearing on the external surface of the malar bone in a line directly above the insertion of the zygomatic arch. This wedge-shaped piece of bone, with its muscular and cutaneous attachments, is strongly forced backward, giving free access to the orbit. The periosteum is incised in a horizontal direction (Arnold H. Knapp). After removal the bone and overlying soft parts are replaced and the latter carefully sutured. The electric circular saw makes quicker and better section of the bone than the chisel.

Osteoma. The ivory exostosis, osteoma eburnea, which is the one most often met with in the orbit, is of very slow and painless growth, and as a rule gives no sign until exophthalmos appears. It is at times well borne until there is decided malposition of the globe, and

[1] About 60 per cent.
[2] About 70 per cent. (Byers).

so insidious is its growth that this is the case even when it has started in the frontal (or ethmoidal) sinus and pushed the orbital wall before it. When it involves or invades the cranial cavity, cephalalgia and other symptoms are apt to set in, and optic neuritis may occur.

Palpation shows a hard, fixed, nodular tumor on the orbital roof or inner wall, as a rule, and a smooth, hard, hemispherical swelling if the seat is in the sinus. But exploration, as stated, is necessary to a correct diagnosis. The etiology is indefinite; rheumatism, gout, syphilis, etc., are of uncertain weight. Trauma has a place. In three fronto-orbital cases of the writer's—two osteoma eburnea—trauma in early life was almost surely a factor.

Treatment. This is extirpation, which is a safe procedure, and the eye, as a rule, can be saved. The same may be said of sinus-orbit cases if the proper method is followed The attempt to remove only the orbital part by attacking the body of the tumor is unsafe, and is not now made. It has been replaced by the method of "subperiosteal enucleation" of H. Knapp, after Maisonneuve. The first step in the operation proper, after reaching the site by incision through the lid, septum orbitæ, etc., is to divide the periosteum over the tumor and peel it off to the basal wall; the latter is then carefully chiselled through close to the tumor, and the latter is grasped and rotated, and then lifted or shelled out. If the tumor be of the sinus-orbit kind—*e. g.*, growing from the frontal—the wall or walls will require chiselling in order to release the growth from its bed. After removal and careful cleansing, etc., the periosteum is reunited by deep sutures, and then the wound closed. Packing or a drainage-tube may be used temporarily, and after-treatment is according to general principles.

Encephalocele is a congenital, fluctuating, nearly always pulsating and compressible tumor, found at the inner-upper part of the front of the orbit, and at times is of large size. It is a hernia of the brain and membranes through a hiatus, which may at times be felt at the site of junction of the frontal, lacrymal, and superior maxillary bones, and is often double. It is extremely rare, and is inoperable. Its diagnosis is only important if the subject lives—the exception.

Sarcoma is the most common growth in the orbit, and occurs more often in children than in adults. It may originate in any intra-orbital tissue, or invade the socket from adjacent parts, sinuses, etc. Sarcoma of the choroid, like its congener, glioma of the retina, may become extra-ocular and then virtually orbital. In some cases the growth forms a large mass, which is covered but partially by the greatly stretched lids—so-called encephaloid or fungus hæmatodes. Even at this stage radical relief may follow exenteration, and this is the more likely if the tumor, though large, be movable and the periorbita unaffected. Prolonged immunity from pain and recrudescence, at least, may be had in seemingly desperate cases; but this applies almost wholly to adults and to mixed forms—*e. g.*, adenosarcoma and fibrosarcoma. The latter, which are encapsulated or

circumscribed and of very slow growth, yield good results to thorough excision. (This holds also in regard to carcinoma, which has occurred only in the lacrymal gland and on the optic nerve.) The consensus of opinion is against operation in orbital sarcoma of children, in whom, as a rule, it is of the small, round-cell, most malignant type, and decidedly if growing from a sinus. It is held to be inoperable in the sense that recurrence is almost certain, and death is hastened rather than retarded. The relief of pain or other special indication or condition may warrant operation.

EXENTERATION (OR EVISCERATION) OF THE ORBIT. Complete exenteration means the removal of all the contents of the socket, including the periosteum to the apex, and also at times ablation of one lid or both lids when involved primarily or in course. The outer canthus is divided by a cut to the bone, the lower lid is drawn down, and an incision is made in the retrotarsal fold to the bony margin from the outer to the inner canthus, and then similarly to the upper orbital rim, passing behind the lacrymal sac if it is healthy.

The closed scissors (strong and curved) are then passed deeply along one or other bony wall and made to sweep, if feasible, around the mass. Traction is made, and the tissues cut as close to the apex as possible; bleeding is stopped by pressure; the periosteum is freed from any remnants by the scissors and sharp curette, and the stump trimmed and treated with 10 per cent. solution of zinc chloride. The outer canthus may be restored by sutures. When oozing has ceased the cavity is cleansed and packed with medicated gauze anointed with sterilized vaseline; a compress is applied over the lids and secured by a bandage. The outer dressings should be replaced on the second day, but the packing may be left four to six days. If the growth is adherent or the periosteum involved, the latter is detached at the margin of the orbit all around and from the walls, so as to be removed entire as far as may be when the tissues at the apex are severed. Any diseased periorbita or patches of bone found are curetted and treated, as well as the stump, with 10 per cent. solution of zinc chloride, or the chloride of zinc paste applied on lint. If the floor of the orbit is carious, it is advisable to explore the antrum, and thorough removal of the contents of the latter is necessary if it has been invaded. It is wiser to sacrifice the lids in part or wholly than to leave any suspicious tissue which may be the nidus of new-growth. The lacrymal gland is always removed; and if the sac is implicated, it should, of course, be cut away and the nasal duct carefully curetted. If the lids are to be sacrificed, the first step is to cut through the skin to the bony rim, and follow this around. Recovery is much quicker and after-treatment simpler if the denuded walls of the orbit are at once lined with Thiersch skin-flaps carefully adapted and supported by packing (Mickulicz and H. Friedenwald). The Thiersch grafts may also be applied later with advantage to the (prepared) granulating surfaces (Busachi). The extreme retraction of the lids is thus averted and a special form of artificial eye may be worn.

Cysts. These may be serous, blood, sebaceous, dermoid, hydatids, echinococci, and cysticerci. They occur mainly in the front of the orbit, are mostly congenital and of slow growth, and are painless unless very large. They may be firm on palpation, but on exposure elastic, if not fluctuating. Hydatids are very rare in America; dermoids are not uncommon. The latter may lie dormant during adolescence, showing as a circumscribed swelling at some point just within the orbital rim, and then at puberty begin to grow. Their contents may be varied, as they are elsewhere. It is well to bear in mind that cysts are often found to dip deeply into the socket and to interpenetrate parts to a degree not suspected before they are explored. And as they may be united to the sheaths of the muscles or of the optic nerve or globe itself, their removal may require careful and deep dissection, with risk to healthy parts. Hence some prefer *obliteration* by incision, evisceration, in hydatids as well as dermoids, etc., and light cauterization of the sac-lining by means of argent. nit. crystals, or sol. tinct. iodine, etc., applying cold to lessen undue reaction, and keeping the mouth open until the walls become fused (Buller, Mathewson, Standish, etc.). Antiseptic injections are used to the same end (Swanzy). Others do and advise extirpation (Berlin, Nettleship, W. S. Bull, etc.).

Exophthalmic Goitre (Parry's Disease, Graves' Disease, Basedow's Disease). This disease, as the name suggests, is marked by proptosis and enlargement of the thyroid gland, which, with tachycardia and tremor, form the so-called cardinal symptoms. The weight of evidence points to the toxic action of abnormal thyroid secretion as the cause of this strange symptom-complex, with a basis of neuropathy.

Etiology. Fully 80 per cent. of the cases are females, and about 33 per cent. occur in the third decade; it is rare early and late in life. The acute form follows fright or other strong emotion, which gives color to the claim that it is primarily a pure neurosis. The usual chronic type is often preceded by a period of care, anxiety, or other nervous strain. Heredity plays a part in so far at least as neurosis is an etiological factor. Central lesions (in the medulla, etc.) involving the sympathetic, which have been regarded as causative, are held by Putnam and others to be most likely secondary. The essence of the disease is some fault in the thyroid (hyperthyrea), which presents a sort of compensatory hypertrophy with perverted secretion (Greenfield, Moebius, Horsley, etc.).

Symptoms. One or other feature of the clinical picture may be lacking, and the evolution of the symptoms is not constant. The onset is generally gradual and the disease chronic. The signs of decided functional disturbance of the nervous system are, as a rule, clearly in evidence.

Tachycardia. Cardiac palpitation and rapid pulse are most often the initial symptoms. The pulse runs from 100 to 140 or more. There are marked throbbing of the carotids and a vascular murmur

over the thyroid. Enlargement of the thyroid is generally patent when the other principal symptoms are present. There is visible pulsation, and a thrill may be felt.

Exophthalmos. The proptosis, which is almost always double and but rarely absent, varies according to the degree of vascular turgescence of the orbit from slight degrees to marked protrusion. So great is the latter at times that the cornea remains constantly more or less exposed. It is then apt to ulcerate, with loss of the eye at the time, or later by secondary glaucoma or septic invasion and panophthalmitis. Hyperplasia of the connective tissue and increase of the orbital fat delay recession of the eye in favorable cases.

There are other ocular symptoms of interest: (*a*) *Dalrymple's sign:* retraction of the upper lid from tonic spasm of Müller's fibres. This causes the notably staring look of such cases by exposing a strip of sclera above the cornea. It occurs without proptosis, and adds to the effect of the latter when present. (*b*) *Stellwag's sign:* infrequent and imperfect winking (*a* and *b* may be due to loss of sensation of the cornea and conjunctiva and of the reflexes (C. Wood). (*c*) *Von Graefe's sign:* loss of the consensual descent of the upper lid in downward movements of the eye. The upper lid lags behind and the sclera may be exposed. In not a few cases this is absent. Insufficiency of convergence from disability of the internal recti is often present (Moebius).

Spontaneous pulsation of the retinal arteries on the disk (O. Becker) also occurs, but is not constant. The retinal arteries may be relatively large; but the fundus is practically normal, and vision is unaffected. Dryness of the eyes is a not uncommon source of discomfort.

The resistance of the body to the electric current is greatly reduced (Wolfenden). Profuse sweating, pulsatory tinnitus, headache, anæmia, and mental depression are common symptoms. Acute cases may reach the acme in a few days or weeks, and recover as quickly, or end in death. As a rule, months may pass before the palpitation and quick pulse are followed by exophthalmos or Dalrymple's sign, etc., and from six months to one, two, or more years before recovery ensues, or the disease may persist.

Treatment. Rest, mental and physical, is important; hydrotherapy and massage are of service; iron and digitalis are often useful in anæmic cases, and the tincture of strophanthus as a cardiac tonic; belladonna, iodine, cod-liver oil, and bromides have proved helpful. Osler, who admits the uncertainty of medicinal treatment and decries veratrum viride and aconite, says, "no measures are so successful as protracted rest in bed with an ice-bag applied continuously by day over the heart, or, what is sometimes more agreeable, over the lower part of the neck and manubrium sterni. I have known the pulse to be reduced in this way from 140 to 90."

The systematic use of the galvanic current has been of distinct service in many cases: a current of from 0.5 to 1.5 milliampères for from one to three minutes on alternate days, the cathode at the

angle of the lower jaw, first one, then the other, with the anode at the back of the neck. Persistent faradization or galvanization of the thyroid gland with strong currents is certainly useful, whatever its mode of action (J. J. Putnam). Thyroidectomy has been of undoubted value despite the large mortality hitherto attending it. Of sympathectomy there have been favorable reports and unfavorable.

Local Treatment. When the cornea is partially exposed, the simplest expedient is the wearing of a light protective compress, at least at night. When the whole cornea is uncovered, tarsorrhaphy should be done; and this failing, the lid margins should be made raw at corresponding points, and be united broadly in the centre or at several points, sutures being inserted sufficiently far from the free edge to hold firmly. This is especially required in progressive exophthalmos, and may save the eyes from destructive ulceration, or arrest the latter so as to prevent utter loss of sight. In a few instances removal of the eye has been necessary to relieve the sufferer from excessive pain due to ulceration, panophthalmitis, etc.

Diseases of the Accessory Sinuses.

Many cases of subacute, if not acute, inflammation of the frontal sinus recover spontaneously, or under intranasal treatment of the exciting rhinitis, etc., without orbital mischief. Orbital periostitis and cellulitis (which see) are sometimes set up by acute suppurative inflammation of one or other of the accessory sinuses, which in turn is likely caused by the invasion of pyogenic organisms in the purulent rhinitis of influenza and ethmoidal and antral empyema, pneumonia, and the exanthemata. Again, *chronic empyema* of the frontal sinus is at times a sequel to the acute form, or is latent in its onset and course and without external sign, complaint being made only of browache and nasal discharge, which, however, are present in ethmoidal empyema. In case of retention from imperfect vent, ulceration of the periosteum and caries and perforation of the bony wall may slowly ensue, and the mucopurulent contents held by the orbital periosteum and fascia, form a tense swelling along and beneath the brow or at its inner half, which fluctuates under pressure. Now and then spontaneous fistula occurs beneath the brow, as in periostitis; but unlike it in that the probe enters the sinus and the discharge is mucoid. Fistula orbitæ may occur also in chronic empyema of the ethmoidal or fronto-ethmoidal cells, which, indeed, sometimes form one cavity with the frontal sins.

In **chronic mucocele of the frontal sinus,** which some embrace under chronic empyema, though only yielding mucus, stenosis of the frontonasal canal and resulting retention and distention cause gradual depression of the floor (orbital roof) and prominence of the anterior bony wall with exophthalmos downward and outward, im-

paired motility, diplopia, ptosis, etc. The smooth swelling which dips back along the orbital roof may feel hard or may yield to firm pressure, and the purulent nasal discharge of empyema is wanting.

Treatment. The general indications are to open the sinus, remove diseased contents, secure permanent free drainage, and then carry out medication, provided the operation does not prove radical.

OPERATION. In acute empyema of the frontal sinus, other treatment failing, an incision is made in the inner third of the eyebrow to the side of the root of the nose, the periosteum separated over a limited area, and a small opening made with drill or chisel through the wall just below the supra-orbital ridge and external to the mesial line, the lining membrane opened, and the cavity irrigated with 1: 40 carbolic, 1: 4000 collyrium of mercuric chloride, etc. The sinus is then explored with the probe, which may carefully be passed into the frontonasal canal to test patency. Protargol, 5 per cent. solution, may then be injected, and medicated gauze inserted. The latter should be changed from day to day, and irrigation and injection practised if there is secretion, the protargol being increased to 10 per cent. or stronger if pus continues and there is no undue reaction. The external wound may be closed after several days if the discharge has ceased; otherwise a short tent, plug, or flanged style may be used, and medication continued as required.

In *chronic mucocele* the opening at the end of the brow in the bony wall should be made sufficiently large to admit a curette or large trocar. After irrigation and careful curetting to remove hyperplasia, mucous polypi, etc., a free opening is made to the infundibulum with trocar, gouge, or burr. Until recently[1] the rule was (as in empyema, which see) then to insert a good-sized rubber drainage-tube, the free ends being fastened above the brow and without the nostril, respectively,[2] systematic use of antiseptic and astringent solutions forming the after-treatment. This may cover three, six, or twelve months. After a time—one or two months—when the lower canal seems smooth and healed, the long rubber or metallic tube is replaced by a short tube, plug, or stylet, and worn in the sinus and hidden by plaster. As a rule, the orbital roof gradually rises, the brow recedes, and the eye resumes its proper place.

In chronic empyema of the frontal sinus freer access to the cavity must be had than is needed in the acute form, or in mucocele proper. The effort is now largely made to render the operation really *radical*. Jansen makes an incision under the brow and cuts away the bony floor, with after-treatment by repeated packing. In Kuhnt's operation the aim is to obliterate the sinus; the whole of the front bony wall is removed and the mucous lining scraped away. To lessen

[1] Some form of "radical" operation is now in vogue.

[2] In twelve cases, under the care of the writer, of chronic mucocele or empyema, with enlargement, requiring operation, this procedure was followed. The treatment lasted from a few weeks to twelve months or longer. In only six cases was it fairly tested, and in these the result was satisfactory.—Canadian Practitioner, May, 1887.

deformity, the periosteum is now left.[1] To the same end, Kipp spares the upper orbital margin. In H. Tilley's modified Kuhnt operation "the cosmetic result in the majority of cases leaves nothing to be desired, and the purulent discharge is permanently cured." Within three weeks from the operation the patient may, as a rule, be discharged. The incision is made from just above the internal palpebral ligament, coming upward and outward just below the line of the eyebrow to a little beyond the junction of the inner and middle thirds of the supra-orbital ridge. After retraction of the periosteum, a good deal, but not the whole, of the anterior wall is removed. After the usual curetting to the periosteum, "all crevices or extensions of the sinus must be searched out with a small curette or sharp spoon." Then a free opening is made of at least one-quarter of an inch lumen into the nose by means of curved burrs or a curette. To effect this and remove diseased ethmoidal cells, a small segment of the nasal process of the frontal bone is cut away. Before packing with antiseptic gauze, the sinus is dried and swabbed out with a solution of zinc chloride, 8 per cent., or pure carbolic acid. The soft parts, including the periosteum, are sutured, except at the inner end where the gauze projects. Later a curved silver wire is inserted, and daily syringing with weak carbolic acid lotion, etc., is practised. Finally, partial obliteration of the sinus is secured by firm external pressure.

Correction of intranasal disease is an important adjunct, if not preliminary; the middle sinus and turbinal require special attention. Grünwald amputates the anterior half of the middle turbinal. It may be advisable (or necessary, Turner) to remove the whole. This is the more important where, as is often the case, combined antral, ethmoidal and frontal sinusitis occur.

Tilley rightly warns against septic osteomyelitis of the frontal bone, which he ascribes (in eight or nine fatal cases collated) to non-provision for free drainage into the nose at the time of the operation. The external wound had been tightly sutured, and septic phlebitis of the diploic veins was set up. The writer had under his care a serious case of burrowing subperiosteal abscesses over the calvarium from a neglected frontal sinusitis.

Another form of operation, the osteoplastic opening of the frontal sinus[2] (S. S. Golovine, after Czerny), which, the author says, "ensures a complete cure, with no depression and scars scarcely visible," is done as follows: A cutaneous incision about 4 cm. long is made along the upper edge of the internal half of the eyebrow, and at its internal extremity another incision is made obliquely to it following the fold of the corrugator muscle of the eyebrow. These two incisions form the letter T placed horizontally, and in depth they reach to the periosteum. The soft tissues forming the upper border of the incision can be detached and lifted up. An arched incision of about 2 cm.

[1] In twenty-five operations, thirteen on one side, and twelve on both sides, the cases were cured in from ten days to two weeks. A number were not disfigured by scars. (Röpke after Kuhnt.)

[2] Archives of Ophthalmology, vol. xxvii., No. 3.

in height is then made through the periosteum, the base of which corresponds to the internal third of the upper orbital ridge. Following the line of incision made in the periosteum, a small groove is hollowed out with a chisel which does not penetrate farther than the diploë. Then, by means of a thin, flat, and very wide chisel, held obliquely, the bone is cut out without danger of entering the skull. This little piece of bone formed from the anterior wall of the sinus can be raised and turned back like a small shutter, the periosteum and soft tissue serving for hinges at its base. The result of this is an opening sufficiently large to admit of complete examination, and enabling one to perform a thorough curettement. A drain is passed through the nose, the small shutter is put back into its primitive place, and the wound is sutured and dressed. Daily lavage through the drain with hydrogen peroxide is the after-treatment.

Golovine has also used *steam* as a caustic injection in empyema of the frontal sinus, passed through a very small trephine-opening at the upper inner angle of the orbit or through existing fistula. "The injection of the steam should last from one-quarter to one-half of a minute, and may be repeated after several minutes." This offers a sure though slow obliteration of the sinus—five or six months' time. Steam may also be used as an adjuvant in operations.

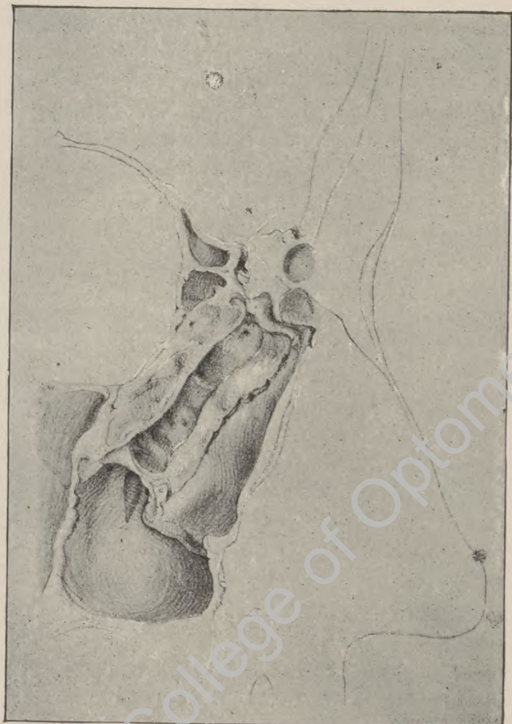
Ethmoidal Sinus. Ethmoidal disease, *per se*, not involving the orbit, is treated intranasally. Secondary orbital periostitis, cellulitis, and abscess require early deep incisions, etc. (See Cellulitis.) In chronic mucocele or empyema of the ethmoid with distention and tumor-like projection into the orbit, a curved incision is made from just under the inner third of the eyebrow to the inner canthus, keeping to the inner upper side of the pulley of the superior oblique (H. Knapp). One may thus explore, curette, make drainway into nose, apply carbolic acid, and insert tent, the wound being then (almost) closed. Afterward irrigation of the ethmoid from in front is done daily or at short intervals, and the tent renewed until the parts heal or intranasal treatment suffices.

Sphenoidal Sinus. The sinus may be reached from the orbit *via* the ethmoidal cells, and this has now and then been nature's pathway of relief; but except in so far as orbital periostitis and cellulitis (which see) are set up by it, the operative treatment of sphenoidal empyema has been almost wholly intranasal. Many cases of sphenoidal empyema have been treated with a high percentage of success and fairly prompt recovery by opening the anterior wall, curetting, and medicating (after ablation of the middle turbinal). In forty-five cases in thirty-four patients the time for cure was never longer than four months (Grünwald).

THE LACRYMAL APPARATUS.

Anatomy. The lacrymal apparatus consists of the lacrymal gland, which secretes the tears, and the lacrymal passages, through the medium of which the tears are drained into the nose. The *lacrymal gland* is an acinous gland in two parts, the larger of which lies in a depression in the bony wall of the upper external angle of the orbit, while the smaller is placed somewhat inferiorly to it, directly beneath the mucous membrane of the fornix.

Fig. 105.



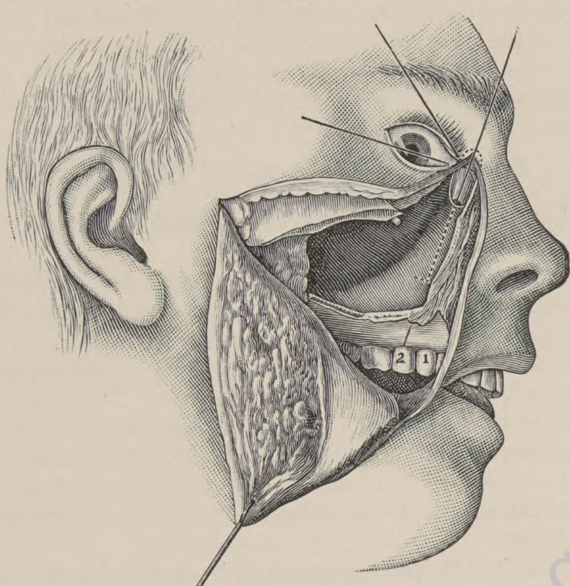
Section of lacrymal sac. (Arlt.)

Both portions of the gland pour out their secretion by a series of small ducts into the upper outer portion of the conjunctival cul-de-sac.

The excretory portion of the lacrymal system consists of the puncta lacrymalia and the canaliculi. The *puncta* are brought into view by everting the lids, and are seen as small papillæ at the inner extremity of the lids at a point where the cartilage terminates. The *canaliculi*, the two fine canals which are the continuations of the puncta, at first run vertically, but soon turn into a horizontal axis and empty

into the lacrymal sac. The *lacrymal sac* (Fig. 105) is situated in a fossa at the inner angle of the orbit and terminates in the lacrymal duct. When distended, the sac is about 12 mm. long and has a diameter of about 6 mm. It is narrowest at its termination into the duct, making this point a favorable one for the development of strictures. The anterior boundary of the sac is formed by the internal lid ligaments, and it is believed that the contraction of these aids the natural elasticity of the walls of the sac in the expulsion of its contents into the nasal duct.

FIG. 106.



Duct seen through the maxillary sinus. The projection which the canal makes in this position forms a cone with its base below directly continuous with the inferior meatus. The apex is directed upward and anteriorly into the lacrymal sac. The axis of the canal is oblique from above downward, from before backward, and from within outward; continued to the dental arch it is seen to terminate at the second molar, while its superior or frontal extremity bisects the superciliary ridge 2 cm. external to the median line; the transverse line traced over the wall of the sinus indicates the point where the canal empties into the inferior meatus. Two pins introduced into the canaliculi show the place of common opening into the sac.

The duct (Fig. 106) varies in length from 12 mm. to 20 mm., and in diameter from 3 mm. to 4 mm. Its general direction is expressed best by a line drawn from the inner canthus to the interval between the second premolar and the first molar tooth of the upper jaw. It terminates in the nasal fossa below the inferior turbinate body. The mucous membrane of the lacrymal apparatus is continuous, but differs somewhat in the different parts, that of the canaliculi being lined with laminated pavement epithelium, and that of the lacrymal sac and duct with a layer of cylindrical epithelium. Owing to the projection

of the mucous membrane at certain points, a series of valves is formed, the largest of which, Hasner's valve, is situated at the inferior termination of the duct. In the duct the mucous membrane is connected by areolar tissue with the periosteum, but is separated from it by a thick venous plexus which has the properties of cavernous tissue.

Though the moistening of the eyeball is chiefly effected by the action of the lacrymal gland, a secretion is poured out by the conjunctiva and its mucous glands as well. It is for this reason that the eye may still be kept moist after removal of the gland.

After the tears have lubricated the eyeball they are sucked into the canaliculi by the action of the lids in forcing the fluid into the inner angle of the eye and through the medium of the ligaments in dilating the sac. Their passage from the sac into the nose is due partly to the weight of the fluid, but chiefly to the elasticity of the sac.

Diseases of the Lacrymal Apparatus.

Dacryoadenitis. Inflammation of the lacrymal gland is very rare. It may be acute or chronic.

In the *acute form* the outer end of the upper lid beneath the brow becomes swollen, red, and tender, with more or less severe pain, and inability to raise the lid, save near the inner canthus. The eyeball may be displaced downward and inward. The focal swelling is not in the lid proper, as in phlegmon of the latter. The adenitis may be symmetrical, so-called lacrymal mumps, and occur with parotitis. It may be caused by trauma, toxic invasion, cold, etc., and it has occurred by metastasis in urethral blennorrhœa, and as a primary syphilitic "symptom." It may end in resolution, suppuration, or the chronic form.

Treatment. Leeches, iced compresses, hydrarg. submur., sudorifics, and laxatives. If the pain, swelling, etc., increase, hot fotus and cataplasms should be used; and on sign of pointing the phlegmon should be opened, preferably in the cul-de-sac, to avoid external fistula.

Chronic adenitis may be subacute in its onset, or merely show slight redness of the upper lid, which is more or less prominent. The swollen gland can be felt below the bony rim, and there is little or no pain or tenderness. Now and then this form is tertiary and gummatous.

Treatment. Topically, ungt. hydrarg., hydrarg. oleat., or tinct. iodin.; internally, potassium or sodium iodide, raising the dose to gr. xxx., or even gr. lx., t. i. d., if well borne, and giving pilocarpine in addition in dose to cause profuse sweating, daily or on alternate days, for ten to fifteen times.

In *suppurative inflammation* of the accessory or palpebral portion of the lacrymal gland there is a tense, tender swelling in the outer upper part of the upper lid, with hyperæmia and localized chemosis

at the site of the small ovoid tumor which projects into the cul-de-sac. This shortly points and opens into the fissure. There is little systemic disturbance.

Treatment. Early application of the lead-and-spirit lotion on compresses may arrest it.[1] If ineffective, hot fomentations are in order.

Hypertrophy of the lacrymal gland is very rare. It may be due to recurrent inflammatory attacks, occurs most often in children, and may be congenital. The gland increases in size very slowly, and there is a notable absence of external signs of inflammation. The tumor is circumscribed, somewhat firm, elastic, and nodulated. In time it may displace the eyeball and limit its movements.

Treatment. Treatment by pot. iodid., especially in adults, in whom the affection may really be a chronic tertiary adenitis, should be pushed, large doses being given if tolerated. External applications of ungt. hydrarg., tinct. iodin., etc., may be used. In genuine hypertrophy, however, operation is generally required, a part or the whole of the gland being removed.

Atrophy of the gland occurs in xerophthalmia. It is said to be secondary to destruction of the lacrymal sac, but this is certainly not a necessary result.

Fistula is caused by injury or abscess. It may be cured by the use of caustic or the cautery point, or by passing a suture with two needles into it and on through the conjunctiva, and tying both ends over a bridge of the latter at the fornix, or by making a counter-opening for drainage into the cul-de-sac by means of a suture as a seton. These steps failing, removal of the gland is indicated. In one case in which the writer did extirpation the tears were escaping on the temporal side of the orbital rim.

Tumors of the lacrymal gland are very rare. Adenoma, adeno-angioma, angioma, epithelioma, enchondroma, osteochondroma, lymphosarcoma, carcinoma, cysts, and dermoid growths have been reported; also miliary tubercle in general miliary tuberculosis. The diagnosis has to be worked out in each case as far as may be, and as an aid a tentative course of potassium or sodium iodide in large doses may be useful, if not decisive. Excision should be done early, so as, if possible, to circumscribe the neoplasm. Calcareous concretions (dacryoliths) sometimes occur in the gland or duct and act as foreign bodies. They show through the conjunctiva, and should be removed by incision.

Dacryops. Cyst of the lacrymal gland appears as a bluish-pink, semitransparent, elastic swelling of varying size showing beneath the

[1] The following anodyne astringent mixture applied on compresses or under an ice-bag is very useful in inflammation of the eyelids, tear-sac, or gland:

Plumb. acetat.,	grs. v.
Acid. acetic. dil.,	♏ v.
Atrop. sulph.,	gr ⅛ to ¼.
Morph. sulph.,	gr. j.
Alcohol.,	ʒj.
Aquæ,	ad ℥j.

conjunctiva at the cul-de-sac, and if large causes prominence of the upper lid. The swelling becomes at once distinctly larger if the subject cries or the gland is stimulated from any cause. It is generally due to closure of an excretory duct with retention of tears and distention of the part of the duct and gland involved. It may be congenital. The treatment aims at securing a permanent opening by incision or the use of a suture, by which fistula in the conjunctiva is produced. Where there is a cystic tumor or hydatids of the gland, the radical operation may be necessary, or treatment of the lining of the sac by tinct. iodin., argent. nit., or acid. carbol. after evacuation, with iced compresses to prevent undue reaction.

Dislocation of the lacrymal gland is almost wholly due to trauma in young subjects, in whom the orbital rim is ill developed. In case of lesion the gland may present in the wound, or there may be an almond-like tumor at the upper and outer part of the eyeball, or a movable tumor under the lid. Gradual luxation may occur.

Treatment. Reposition may be possible, and if not, excision may be required; and, again, interference may not be called for.

Epiphora. Normally there is only sufficient lacrymal secretion to keep the eye moist, and there is no stream of fluid passing through the puncta. A flow occurs only where there is hypersecretion, so-called lacrymation. When there is much lacrymation the natural lacrymal passages are inadequate, and the tears collect in the lacus or they overflow—a condition termed epiphora. Defective drainage does not account for the surplus often present; the gland frequently acts in case of stricture of the canaliculus or duct as if there were a foreign body to be swept away. A most marked case of epiphora of years' standing was cured in two days by simply opening the canaliculus into the sac. Epiphora may result from hyperæmia or inflammation of the intra-ocular tissues or of the conjunctiva, cornea, and lid-edges; injuries of or foreign bodies on the eye or beneath the eyelids, when it will be sudden; malposition of the lid-edges or of the puncta, and atresia of the puncta or canaliculus; mucocele (catarrh of the sac with dilatation), stricture of the nasal duct and lacrymal fistula; also rhinitis or defect in the turbinals. Young subjects with chronic coryza and boggy turbinals often have epiphora, which ceases when the nasal trouble is cured. Lacrymation may also be due to reflex irritation, mainly through the medium of the fifth nerve; sympathy with the fellow eye and emotional causes are well known. Eyestrain as a factor is to be borne in mind. Some cases of habit lacrymation are due to it.

A rare cause of epiphora is blocking of the canaliculus by concretions of Streptococcus Foersteri. It also arises from notching of the lower lid, and from flaccidity of the lids due to loss of tone of the orbicularis, and in paralysis of the latter it is most annoying, the interspace between the globe and lower lid being filled with tears which often overflow—lagophthalmos.

As other and serious lacrymal troubles are generally preceded by epiphora, its cause should be early found out and dealt with. This may require some study. Two or more of the conditions cited may coexist, either of which would suffice—*e. g.*, chronic conjunctivitis and contracted or everted punctum, rhinitis and associated conjunctivitis, optical defect, blepharitis, etc.

Puncta and Canaliculus. Closure of the puncta (atresia) is rare It may be congenital or due to shrinking after inflammation or injury, malposition, etc. The punctum should be reopened and stretched a few times by a silver pin, blunt needle, or fine sound. Small, even minute puncta may not cause epiphora, but it does occur when their contraction arises from thickening of the mucous lining and hypertrophy of the sphincter fibres, due to chronic or recurrent conjunctivitis, blepharitis, etc. The puncta then resist stretching by the fine sound, and grip it like solid rubber—too tightly for mere spasm. If after several forcible dilatations there is but little change, the inner wall of the punctum should be snipped vertically with fine scissors, to make a permanent patulous slit, and in atresia proper this has to be done if a trial of stretching fails. The treatment of inversion of the punctum is that of the entropion, etc., which causes it.

In eversion of the punctum due to sagging of the lower lid or to slight ectropion, the canaliculus should be slit and its inner lip and a segment of conjunctiva cut away, making a triangular raw surface with base out. This in healing often corrects the faulty position and the epiphora. Reposition of the everted punctum caused by eczema, etc., of the lid generally follows cure of the latter. It may, however, be necessary to open the canaliculus well down on its inner wall with scissors. In paralysis of the orbicularis (facial) more has to be done, and tarsorrhaphy (see) is needed, and the better to raise and tighten the limp lower lid its inner end is made raw just below the canaliculus, and is stitched to a raw spot at the junction of the upper lid and nose, or instead two small flaps are made and stitched together (H. Noyes).

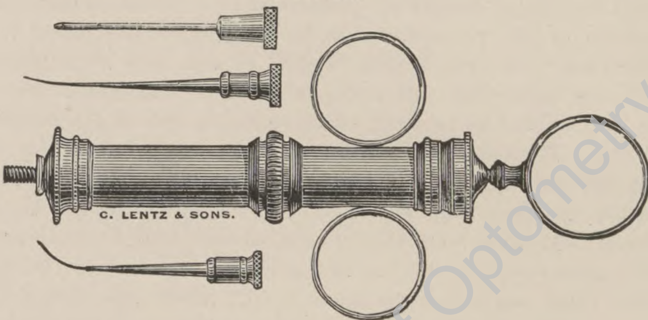
Stenosis of the canaliculi, unless congenital, is as a rule at the inner end next the sac, and care is needed in dilating the stricture to avoid making a false passage. The lid being made taut by traction with the finger-tip on the malar process, a small probe, No. 2 B. or 3 T.,[1] is passed vertically into the punctum, then horizontally along the canaliculus in the line of least resistance, at the roof, as a rule, with a rotary motion, if needed, to avoid piercing the mucous folds. Then, if a slight push inward or downward causes a distinct wrinkling of the skin at the inner canthus, the sac has not been entered, and gentle pressure should be used to force the constriction. The outer wall of the sac will often yield enough before the probe to mislead the inexperienced operator, and the point being forced

[1] Theobald's set is of 16 sizes: No. 1 has a diameter of 0.25 mm.; No. 2, of 0.50 mm.; and so on to No. 16, which is 4 mm. Bowman's set is of 6 sizes: "reaching from a fine hair probe, No. 1, to one of one-twentieth of an inch in diameter, No. 6."

downward, passes through the wall itself, and, it may be, between the duct and the bony canal. To facilitate dilatation of the stricture, a boring motion may be tried and a smaller probe or special sound used (as Theobald's new model). The stricture should be stretched to take No. 4 or 5 B. or T., which should be passed every day or two for at least a few times.

Rarely the canaliculus is the seat of a fungus (streptothrix), and presents an ovoid swelling, with viscid discharge from the patulous punctum; the caruncle and fold are hyperæmic and the eye irritable and watery. The fungous mass (dacryolith) may be expelled through the nasal duct by syringing per punctum. In this way in one case of the writer's, with swelling of the sac and seeming incipient cystitis, a globular mass of the size of a large currant was forced out whole by the anterior naris, and the one flushing—with liq. hydrarg. perchlor. dil.—sufficed. In another the canaliculus had to be opened; the lumen was found greatly enlarged, and the lining much inflamed. The curette had to be used. with after-treatment by sol. hydrarg. perchlor. and protargol.

FIG. 107.



Anel's lacrymal syringe.

The Duct. The anatomy of the nasal duct goes far to show the ease with which some fault may arise to cause epiphora, to wit, the continuity of its lining with that of the nasal mucous membrane, and of its submucous venous plexus with the erectile cavernous tissue of the turbinals.

There is also the fact of the lacrymal tube lying in a bony canal, the periosteal lining of which may play some part—*e. g.*, in scrofula, syphilis, rheumatism, etc. Engorgement of the submucous cavernosa, easily set up, and catarrhal inflammation of the mucous lining by extension may singly or together close the lumen and cause epiphora. Folds in the mucous membrane at the top of the duct where it is narrow, and at its nasal end, if not in the middle, add to the mechanical effect of inflammation and turgescence. Recurrent inflammation set up by nasal disease may lead to structural changes in the mucous membrane of the duct, infiltration, swelling, hypertrophy, and induration, and also ulceration with dense cicatrix, and

thus cause partial stenosis or complete stricture, with secondary implication of the sac. (See Mucocele.)

One can test fairly well the patency of the nasal duct by syringing, the blunt fine tip of a hypodermic or of a lacrymal syringe (Anel's) (Fig. 107) being passed into the sac; fluid injected under gentle pressure should find vent by the anterior or posterior naris. To give the test value, the canaliculus should, of course, be patent, and the other punctum should be closed by pressure. To pave the way for probes, and also to test whether or not a constriction found is due to organic changes—*e. g.*, stricture—or only to vascular turgescence, the injection into the duct of sol. adrenalin chloride 1.5 : 8000, with sol. cocaine, 5 per cent., is useful.

To explore the nasal duct, a No. 4 B. or T. probe is passed through the canaliculus (as directed) and pushed on until the finger-nail resistance of the lacrymal bone at the inner wall of the sac is felt. Then, the point of the probe being kept against the inner wall of the sac, its axis is changed to the vertical, and pressure is made downward and slightly backward, to coincide with a line from the inner end of the caruncle to the point of junction of the ala and cheek. A No. 4 Bowman probe is safer to begin with than a No. 2, and the length as well as the line of direction of the canal should be kept in mind, else a stricture at its lower end may not be passed —a mistake too often made. It is a good rule to gauge the buried part of the probe; it should reach from the caruncle to the edge of the ala.

Treatment. The treatment of simple epiphora due to catarrhal conditions in the duct consists in a short course (of ten to twenty sittings in four to six weeks) of careful probing of the duct with the largest sound the unslit punctum or canaliculus will take, 6 Bowman or 5 Theobald, and gentle syringing with mild astringent solutions—*e. g.*, 1 gr. sol. zinc sulph., zinc chloride, etc. This, with attention to any conjunctivitis, blepharitis, nasal trouble, or eyestrain, will tide not a few over one, two, or three years, and in cases of relapse a few visits may suffice to secure a like respite. But should a fair trial of such probes early fail, the canaliculus should be split (see Bowman's operation) that larger ones may be used. In some cases a short treatment every six months may be needed to correct narrowing, due mainly to chronic or recurrent rhinitis.

Although the epiphora of conjunctivitis, blepharitis, keratitis, etc., is incidental, one can at times give relief and promote cure by attention to a contracted or displaced punctum, or by enlarging the lumen of the canaliculus and duct by the use of probes. This course should not be neglected in some forms of chronic or recurrent keratitis in young subjects as well as in adults, in which treatment also of any nasal affection is a necessary adjunct.

Slitting the Canaliculus (Bowman's Operation). The punctum if contracted is first stretched with the fine conical sound, and a 2 per cent. to 5 per cent. solution of cocaine is then injected into

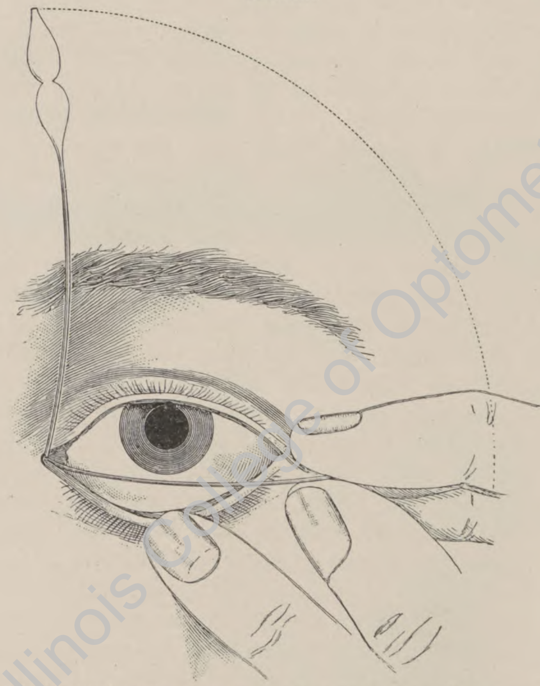
the canaliculus, and the patency of the latter ensured by passing a No. 4 B. or T. into the sac. If this be not done, a false passage is apt to be made above a stricture in the canaliculus. The lower lid being made taut by traction toward the malar process, and slightly everted, the blunt-tipped or probe-pointed knife (Fig. 108) is passed

FIG. 108.

Weber's canaliculus knife.

into the punctum vertically; the handle is then dipped, and the knife with the cutting edge up and in is pushed on past the caruncle until the sac is entered, when the handle is brought again to the vertical, the upper inner wall of the canaliculus being divided to the caruncle, or to the sac, as desired. In cases of mucocele, where syringing or irrigation and the use of larger probes will follow, a freer opening

FIG. 109.



Method of inserting Bowman's probe. (NORRIS and OLIVER.)

is required, and the sac should be entered and cut upward. Care should always be taken not to injure the floor of the canaliculus, which would cause risk (not imaginary) of fusion of the walls; traumatic stricture is a serious bar to a good result. (Figs. 109 and

110.) Again, if a false passage is made over a stricture in the canaliculus, failure is courted, for a few days after the course of probing stops, the new canal may contract or close.

Some prefer, as does the writer, to use fine but not sharp-pointed curved scissors in lieu of the knife when the sac wall has not to be opened. With the lower lid everted and made taut, one point being pushed into the canaliculus with convexity toward the eyeball, a single snip makes a curved cut on the inner wall, which is hidden from view—a point in its favor with females. The raw lips if kept apart a few days heal separately The majority of operators choose the lower canaliculus, but some always slit the upper. In this case the upper lid is drawn tightly toward the brow, care being taken not to cut the front wall (skin). Some open both canals (rarely needed), while others—not a few—will not cut either, and use only such probes in the duct as can be passed through the intact canaliculi.

FIG. 110.

Bowman's probe in position.

FIG. 111.

Mucocele.

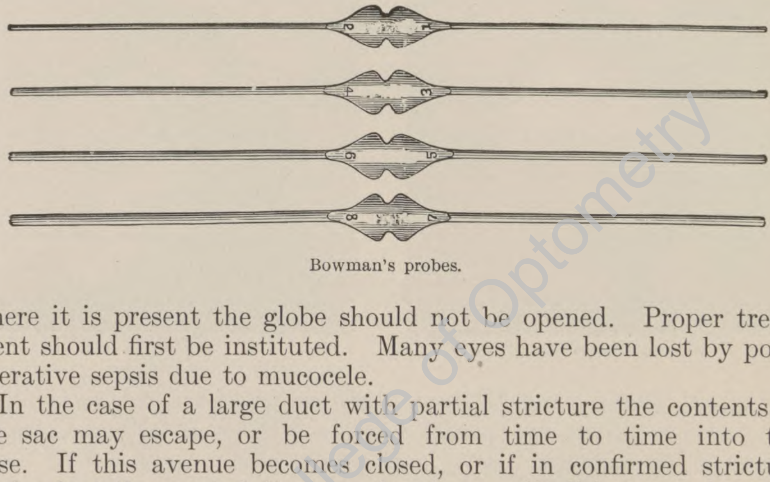
But more violence may be done by forcing probes than by a clean cut; besides, Bowman's operation does not interfere with the normal action of the drainage system. It also permits of the use of large probes, tends to ward off acute cystitis, and enables patients to use the syringe themselves in the after-treatment.

Mucocele. This is a subacute cystitis of the lacrymal sac in which the latter becomes distended by the pent-up secretion from its inflamed lining membrane, together with tears. It is secondary, as a rule, to stricture of the nasal duct, and this, in turn, to recurrent or chronic rhinitis. Fortunately, it is often one-sided, as indeed nasal deformity or disease proves to be. Mucocele mostly develops insidiously, as does the nasal stricture, and there is often simple epiphora of varying degree due to the latter, for months, if not years, before the cystitis supervenes. The retention of tears and the presence of organisms in the sac tend to light up hyperæmia of its lining, with secretion first of mucus and later of mucopus, and gradual disten-

tion ensues. The subject then finds that there is a doughy swelling at the inner canthus which yields discharge, and, getting relief from repeated emptying of the sac by means of the finger-tip, continues the practice. Inspection and palpation show the contrast between the two sides, the lacrymal crest and mouth of the duct being readily felt on the sound side. If there is a lumpy feeling after emptying there is likely much thickening of the lining, or a polypus. (Fig. 111.)

Mucocele may persist for years, causing annoyance only by the epiphora and the blurring of sight by flecks of discharge from the sac or conjunctiva. Conjunctivitis and blepharitis are often present. If absent and the punctum acting, epiphora itself may be wanting. There is, however, always the risk of acute inflammation, and subacute attacks are not infrequent. Besides, the contents of the sac being charged with organisms, infective ulcer of the cornea may occur from slight abrasion, etc., and the eye be lost. The subjects of mucocele should always be warned of their danger, and

FIG. 112.



Bowman's probes.

where it is present the globe should not be opened. Proper treatment should first be instituted. Many eyes have been lost by post-operative sepsis due to mucocele.

In the case of a large duct with partial stricture the contents of the sac may escape, or be forced from time to time into the nose. If this avenue becomes closed, or if in confirmed stricture of the duct the wonted discharge by the canaliculi is stopped, owing to swelling, the tension of the sac may lead to acute cystitis or to great stretching with a capacity of a drachm or more. The real size often does not show because the sac dips backward. As a final stage of neglected mucocele atrophy of the thinned mucous lining of the greatly enlarged sac may occur, and the latter become a mere cistern for the tears (the "Atony and Dropsy" of Fuchs).

Treatment. This consists mainly in cure of the stricture by the systematic use of probes. The first step in a course of probing in mucocele is to slit the canaliculus (page 221), and then to wash out the sac by syringing with a 4 per cent. sol. acid. boric., or sol. hydrarg. perchlor., 1 : 3000. It is unsafe to probe the duct until this is done, unless it holds merely mucus and tears, and even then it is unwise,

for the sac being a favorable culture bed, organisms may gain access to the tissues around through an abrasion or false passage, and set up septic inflammation. Orbital cellulitis, optic neuritis, atrophy, and blindness may result. In syringing after probing, no force should be used, else weak spots in the sac wall may give way before vent is had by the duct. Where irrigation is used instead of syringing, the sac and duct being flushed with a quantity of fluid on the siphon plan, a method preferred by some, the patency of the duct should be tested before pressure is made.

The object of probing is to restore the normal calibre of the nasal duct at the strictured points, with a view to present drainage and future patency. Views differ as to the best method: some will not slit the canaliculus, others will not use larger probes than 6 Bowman. Not to do the Bowman operation and to continue using probes which will pass the intact canaliculus, ignores cases for which the best cannot be done unless large probes are used, and such cases are not few. Large nasal ducts are often found in mucocele, and with one or more ring-like ledges having a lumen of 1.5 mm. to 2 mm., which take the largest Bowman probe. (Fig. 112.) Again, the bony duct in the same subject may differ in size; on the side of the large canal there may be mucocele, and on the other simple stricture. The latter will probably yield to ordinary probes, which would be of little or no use in the former; hence the eclectic plan is a safe rule and the best in the end. Gauge the probe to the duct, and try large ones when smaller fail.[1]

After injecting 5 per cent. of cocaine and 1: 5000 sol. adrenalin, a No. 4 B. or 5 T. should be tried first; and if it fits tightly, it should be left in a few minutes; if not, higher numbers are at once tried, and on the next visit a size larger than that last used.

Probing should be done on alternate days for two or three weeks, and then twice a week for a month, or until the sac has ceased to secrete, when a few visits at intervals of ten days may suffice. If, after the first two or three visits, probing causes only transient pain, one may then safely use the largest size passed under firm pressure, the probes being left in fifteen to thirty minutes. But if the pain lasts several hours in spite of cold compresses, and there is aching in the bone the next day, there is risk of periostitis, and a rest of several days should be given. Syringing or irrigation should, however, be kept up steadily, daily if possible, for its astringent and curative effect, not only on sac and duct proper, but the lower turbinal. In young or very nervous subjects, and where one is asked to do the most in the least time (and this occurs too often), general anæsthesia is required. This allows rapid and, where needed, forced dilatation, and the insertion of the largest styles taken. This plan

[1] Theobald uses his No. 16 "in about two-thirds of all cases requiring probing, including those in children as well as adults, and the cases in which No. 13 may not be used with advantage are extremely rare." He reports the exceptions rare to permanent cure where this line is faithfully followed.

suits in young subjects, the style being left in a few days at least and in older ones it can be raised daily, if needful, to empty or flush the sac. (See Styles.) Electrolysis has been tried in order to get quicker and more lasting effect upon strictures than that by simple probing or the use of styles. The negative pole is connected with the probe *in situ*, and the sponge of the positive is applied to the cheek or neck, the strength of the current not to exceed three milliampères, and time of sittings from two to five minutes. The method, which is worthy of trial, has met with some favor, though not largely used, and it is still *sub judice*. Cataphoresis is also on trial to a limited extent. Solution of nitrate of silver and protargol have been used in purulent blennorrhœa of the sac and duct. Less often than formerly strictures of the duct are first freely divided to the bone by the Stilling or other knife, and at once, or shortly, and from time to time, the Weber biconical or other large sound is passed. An after-course of syringing may not be necessary when the sac is not large and the secretion scant and free from pus; but, as a rule, it is a most useful adjunct. In view of the return of the stricture and of the blennorrhœa in not a few cases of mucocele, even after careful treatment, some are content merely to slit the canaliculus, dispense with probing, and rely upon syringing of the sac with astringents, etc., at home. The so-called Berlin lacrymal syringe, with bulb and two points, hard rubber and metal, is a useful form for this purpose. Some excise a part only of the sac wall, and curette the rest, or treat it topically with sol. argent. nit., etc., and shortly allow the wound in the skin to close. This line of treatment suits some cases of trachoma of the sac or of polypus, which give a pulpy sensation to the finger-tip after emptying the sac, owing to a marked thickening, etc., of the mucous lining. (See Fistula.) In lieu of ordinary extirpation, the sac may be entered and treated from below by removing the anterior part of the inferior turbinate and the turbinal crest with a gouge, etc. (Passow).

Mucocele in Infants. The short and patent nasal duct of most infants gives organisms easy access to the sac, which forms a good nidus. Acute or subacute dacryocystitis may thus cause purulent rhinitis shortly after birth, as early as one week, and the diagnosis is pretty clear. But blennorrhœa of the sac, which more often results, is a mild process, and the flat nasal bridge and relative prominence and width of the inner canthus tend to mask the real mischief, mucocele. Hence, in very young subjects without real or with but slight conjunctivitis this is at times mistakenly held to be the cause of the purulent or mucopurulent discharge found about the eyes now and then during the day, or gluing the lids in the morning. The astringent collyrium as generally prescribed is, as a rule, ineffective. The cul-de-sac and inner canthus region should be filled with 1 gr. sol. zinci chlorid. or acetat., or 1 : 12,000 hydrarg. perchlorid., and short, careful massage of the sac done—this daily or twice a day.

In persistent or recurrent mucocele of infants periodic medication

of the sac by syringing suffices, as a rule, and should be tried. A hypodermic syringe answers well, the blunted and smoothed needle being passed into the canaliculus, and sol. zinci chlorid., zinci sulph., plumb. acetat. of 0.5 per cent. strength, sol. hydrarg. perchlor. 1:3000 or 1 : 4000, slowly injected. If the fluid does not pass into the nose, one should make sure that the tip is in the sac before diagnosing stricture. If the sac contains pus, or if there be mucopus after a few injections made at intervals of two or three days, sol. protargol. 10 per cent. should be used, and 20 per cent. if this fails after one or two trials. Rhinitis should always be looked for, and be treated (as well as in older subjects), else the treatment may prove futile, and this rule holds in all young subjects, upon whom one should be slow to use instruments. In not a few cases the free end of the duct is blocked, as it may be in adults, by a fold of mucous membrane which interferes with drainage, although yielding under the pressure of the syringe. This emphasizes the need of attention to the nasal passages in lacrymal cases. Proper treatment of the inferior turbinals often cures marked simple epiphora, and is an effective adjunct to medication of the sac in case of mucocele. In very young subjects there may be stricture of the duct with (and without) mucocele, which requires slitting of the canaliculus and the use of probes, etc., as in adults.

Dacryocystitis. Acute inflammation of the lacrymal sac is generally a sequel to mucocele, although now and then it lights up primarily as a complication of acute coryza (influenza, la grippe), especially in young infants; also of erysipelas. In scrofulous or syphilitic subjects periostitis or osteitis in the lacrymal region also causes acute or subacute cystitis. After exposure or in the course of rhinitis, etc., the subject of mucocele finds that pressure on the inner canthus does not disperse the doughy swelling as usual, but that the latter has become hard, tender, and tumor-like. Quickly pain, often intense and due to tension, sets in with inflammatory œdema, which in marked cases closes the eye and extends to the cheek and over the nasal bridge, involving the lids of the opposite side. With the canaliculus and duct closed, there is now a virtual abscess, which if unrelieved within a few days points and opens below the internal tarsal ligament, the pain, swelling, etc., quickly subsiding. The rapid onset and smooth glistening skin with bright blush have time and again led to a diagnosis of erysipelas, but the history of epiphora and mucocele, and the intense localized pain and exquisite tenderness at the site of the sac, should give the clue. Generally the breach soon closes, and there is once more mucocele or simple cystitis, which may later lapse again into the acute form. Very rarely the sac resumes the normal condition. Sometimes the opening in the skin persists, giving vent to the tears and mucopus (fistula lacrymalis), a sure result in bone disease, which may be the effect as well as the cause, as already stated, of acute cystitis. In a few cases there is a dissecting infiltration of the skin down and

out from the inner canthus, forming a doughy broad ridge for even an inch, like a fresh keloid cicatrix. In other cases, and rarely, the fistula contracts until there is but a very fine opening in healthy skin through which tears only can ooze—capillary fistula.

Treatment. In mucocele with incipient acute cystitis the rule is promptly to slit the canaliculus into the sac when great swelling of the parts does not prevent it. The tension of the sac is thus relieved, vent given, and pain relieved. Other steps are gentle syringing with warm boric acid sol. 3 per cent., or 1: 8000 mercuric chloride sol., if not too painful; also the injection of 10 per cent. sol. protargol, and the use of dressings wet with atropine, lead-and-spirit lotion, over which a small ice-bag is placed, or a piece of ice in a small gauze sling; calomel, Seidlitz powders, and pulv. Dover., with other anodynes, may prove useful. If cold is not grateful, hot fotus now and then should be used. The canaliculus should be kept open and the sac washed out daily with warm boric acid sol. 3 per cent., or 1: 8000 to 1: 4000 mercuric chloride, and protargol 10 per cent. to 20 per cent. be injected if pus continues. In primary acute attacks in infants, and sometimes in adults, when of a mild type, palliative treatment by iced compresses, etc., may suffice without slitting. The sac should be syringed with warm boric acid sol. through the dilated canaliculus, and 10 per cent. protargol or argentamine then injected. If the case is not seen until too late to reach the puncta, and the inflammation is progressing, one should anticipate rupture through the skin by cutting straight into the sac below the palpebral ligament. After gently syringing with boric acid sol., hydrarg. perchlorid., etc., aseptic moist dressings should be applied and hot fotus used. Irrigation should be practised daily. The canaliculus should be slit into the sac as soon as feasible, for vent given in this way promotes thorough cleansing and the healing of the skin wound, and expedites the necessary resort to systematic probing for the cure of the stricture. The use of probes should be begun as soon as the active stage has passed.

Fistula Lacrymalis. This often needs no special treatment, and heals when the canaliculus is opened, or the patency of the duct is restored by probing. Now and then fungous tissue within and at the mouth of the canal, or, again, the smooth lining of an old fistula, requires to be removed by caustic, cautery, curette, or scissors, to ensure healing. But if the sac is large and secreting pus, it should be opened by enlarging the fistulous canal freely, and the diseased lining swabbed with sol. argent. nit. 2 per cent. to 10 per cent., zinc. chlorid. 5 per cent., or tinct. iodin., or lightly touched with argent. nit. 50 per cent., in the form of a bead fused on the end of a probe; or if trachomatous, curetted, irrigated with sol. hydrarg. perchlor. 1 : 1000, dried, treated with vaseline, and packed, and cold dressings put on. One or other of the above may be needed several times at short intervals before the discharge ceases or so abates as to allow closure of the wound. A pressure pad then promotes healing,

vent being had through the canaliculus already opened. An after-course of probing is useful. In the event of imperfect healing of the fistula or a fresh outbreak of cystitis, caries in the duct or of the lacrymal bone, with internal fistula, will likely be found with the probe, a fair indication for sacrifice of the sac, with careful scraping of the carious spots, etc. In this condition, however, forced dilatation of the strictured duct has been found by Theobald to cure many cases, and this method should, therefore, have precedence.

Rarely a fistula exists at the inner canthus near the sac, but unconnected with the latter and leading to a carious spot at or within the inner margin of the orbit, or even into the anterior ethmoid cells, as shown by the probe. Again, in mucocele of the anterior ethmoid cells there may be a swelling just behind and above the lacrymal sac which simulates mucocele of the latter. The absence of lacrymation and escape of the discharge per punctum on pressure, and the passage of fluid into the nose on syringing, together with deep palpation, should distinguish the condition.

Styles. These may be hollow or solid, and are preferably of soft virgin silver or aluminum; lead wire often is used; some employ gold, and others hard rubber. The crook should be sufficiently long to reach nearly to the punctum, lying in the canaliculus, so as just to avoid touching the cornea in extreme adduction, and the stem should about reach the floor of the inferior nasal meatus. They should be made perfectly smooth, so as not to tear the mucous membrane. The style should be as large as will tightly fit the duct under the use of cocaine and adrenalin, and not smaller than No. 4 B. At short intervals larger sizes can be put in as stricture or hypertrophy yields, and when of large calibre that of the crook should be reduced. If too short in crook and stem, the style is apt to slip down, especially if heavy, as of lead, and the crook become encysted in the sac wall. It will then act as a foreign body, and will need to be cut out of its bed. Hollow styles slipped into place over a probe already passed (Bickerton) are useful, but prominence of the brow may be a bar. Where there is mucocele or blennorrhœa the styles should be drawn up or removed daily and the sac irrigated with an astringent solution. Patients who learn to insert them themselves have the best results. In organic stricture styles should be worn if possible for six months, and if used for other cases are to be worn four to six or eight weeks, a trial respite for two or three days being given now and then. Unfortunately, in many cases styles are not tolerated, and in not a few the tendency to the formation of granulations prevents their use for more than a few days at a time.

With the proviso that a fair trial of probing, styles, syringing, etc., treatment of nasal passages, etc., has been given, the indications for extirpation or obliteration of the sac are: bony stricture of the nasal duct, with constant epiphora, with or without mucocele; recurrent stricture of the duct and purulent blennorrhœa; persistent muco-

cele, with repeated attacks of acute inflammation—phlegmonous; persistent fistula, with inveterate mucocele, etc.; marked dropsy of the sac.

C. Holmes, who urges and practises extirpation of the lacrymal gland and sac together, observes the following indications for the latter:

1. Imperative operations for cataract, glaucoma, etc., in the presence of blennorrhœa, etc.
2. In patients who cannot devote the time, or submit to treatment by probing, etc.
3. In all cases where conservative treatment has failed to cure within a reasonable time.

Extirpation. The canaliculi are destroyed by the glowing wire. The lower end of the nasal duct is sealed by packing gauze under the inferior turbinal. A careful incision is made from a point just beneath the centre of the lower border of the tendo oculi, dissecting obliquely inward and backward and in line with the fibres of the orbicularis, exposing the sac up to its dome. The lips of the wound being retracted and the bleeding stopped; the sac is then freed by a blunt hook and cut off at the edge of the orbit. If this is not feasible, the sac should be cut out piecemeal, and any carious bone should be scraped, to secure, if possible, healing by first intention. The duct is now curetted and the parts irrigated. The wound is closed by sutures and the dressings applied, pressure being made by a compress and strapping. The stitches may be removed in three or four days. The nasal passage should be kept as aseptic as possible until scar tissue has blocked the way to the soft parts at the top of the duct.

Obliteration. To this end, caustic and the cautery are used, to cause sloughing of the mucous lining of the upper end of the duct and of the sac, and fusion of its walls. The canaliculi should first be sealed by the careful use of caustic or the fine glowing wire. Usually the sac is cut into just below the palpebral ligament, or a fistula is enlarged, bleeding is stopped, and the sac cleansed; oil or vaseline is applied around the opening and just within; the cut is made to gape, and argent. nit., in the form of small beads fused on the end of probes or crystals held in fine forceps, is freely applied within the sac; similarly potassa cum calce or acid. nitric. on a small tuft of absorbent cotton may be used; also acid nitrate of mercury, zinc chloride paste (25 per cent.), and potassa fusa.

The sac cavity may also be reached by cutting through the floor of the canaliculi (C. R. Agnew), and caustics, etc., may be safely applied, but care is needed to spare the conjunctiva and to avoid the formation of a scar. The thermocautery and galvano-cautery may also be used to destroy the sac, but the whole sac lining cannot be so readily reached as with diffusible caustics. In either case, cold compresses are applied for a few hours to limit reaction, and then warm poultices of slippery-elm in small muslin bag for several days.

The eschars should be removed as soon as separable, and a pressure compress applied over vaseline dressing, to promote healing. This covers two or three weeks from the start; extirpation requires only one.

The following neoplasms have been found in the lacrymal sac: sarcoma, epithelioma, angioma, fibroma cavernosa, rhinoscleroma; they are very rare except by extension in the case of the two first.

Epiphora frequently persists after destroying or removing the sac, or it often occurs from slight exciting causes: sacrifice of the gland at the same time is, therefore, urged by some as a rule of practice, and the more because under strict asepsis extirpation is a safe operation.[1] But lacrymation is not always a sequel to loss of the sac, and some seemingly intractable cases under the usual treatment are cured by forcible dilatation of the strictured duct with large-sized probes (Theobald). Hence the gland may well be spared for a time until this procedure has been tried.

Extirpation of the Lacrymal Gland. The parts being surgically clean, a cut is made just below the outer half of the eyebrow to the margin of the orbit, exposing the septum orbitæ, which is then divided close to the bone, with just sufficient edging to hold sutures. Fatty tissue may present, but should be left. The lower edge of the gland does not, as a rule, reach to the bony rim, but entire removal should be aimed at, and can with care be effected by scissors, hooks, etc., and without injuring the levator palpebræ or external rectus. All bleeding should be stopped. The palpebral portion (inferior, secondary or accessory) need not be removed. Careful stitching of the skin wound, the fascia being first united by buried catgut, and aseptic dressings under compress, ensure prompt healing and but little after-sign.

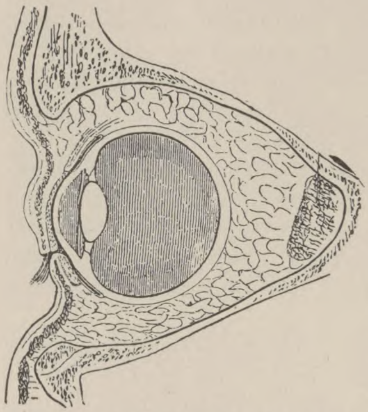
THE EYELIDS.

Anatomy. The eyelids are two folds of skin which cover the eyeball, and by their membranous attachments close in the orbital cavity. The lids owe their form and stiffness to cartilages or *tarsi*, as they are called (Fig. 113); these are the framework of the lid, and when they have been distorted as the result of disease or accident, interfere seriously with the lid performing its proper functions. The cartilages are covered externally by the skin and the orbicular muscle externally by the conjunctiva. The borders of the lids are fringed with short hairs, the lashes, or *cilia*. These are directed forward and are more numerous upon the upper lid. As is shown by the accompanying illustration (Fig. 114), there is a depression or *sulcus* just below *j*, which represents the opening of a sebaceous gland. The cilia are seen at *h*, with modified sweat and Zeiss glands about them. Beneath the skin lie the transversely divided bundles of fibres of

[1] "Primary Union in Eighteen Cases out of Nineteen." C. Holmes.

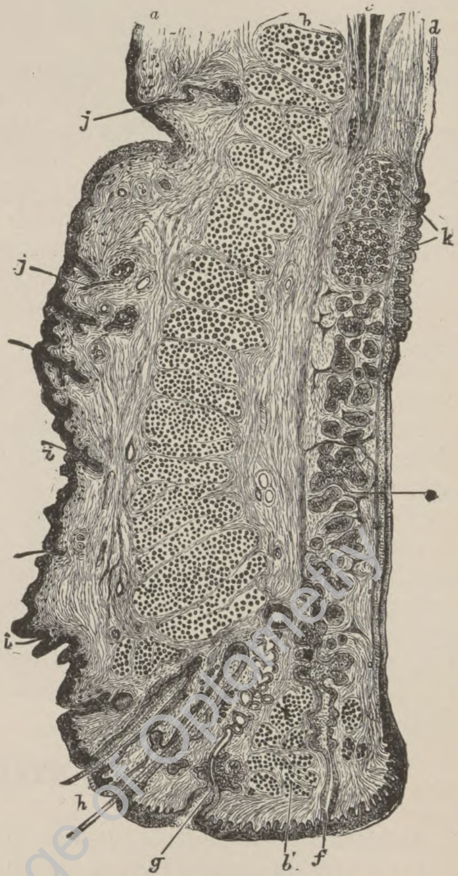
the orbicularis (*b*), of which those placed internally (*b'*) form *the musculus ciliaris Riolani*. The posterior part of the lid is covered

Fig. 113.



Relative positions and sizes of eyelids. (Merkel.)

Fig. 114.



Vertical section through upper eyelid. (Shäfer.)

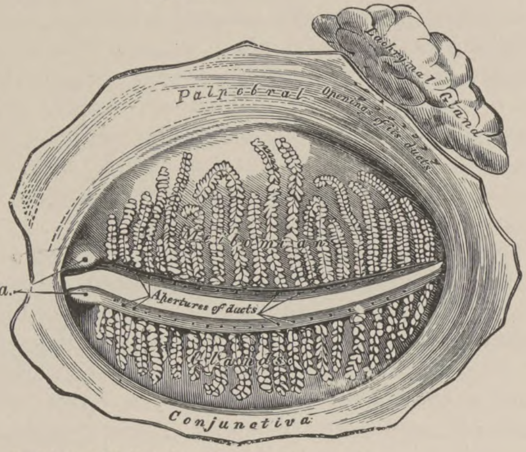
by conjunctiva, which is closely adherent to the tarsus. The *Meibomian glands* have their orifices in front of the posterior edge of the lid; above them lie the *mucous glands* (*k*), and still higher *Müller's muscle* and *the levator* of the lid. The *Meibomian glands* (Fig. 115) are modified sebaceous glands, and secrete a sebum which bathes the margin of the lids and prevents overflow of the tears. The closure of the lids is effected by the *orbicularis muscle.* The fibres of this muscle form more or less of a sphincter, extend into the subcutaneous tissues surrounding the lid, and are inserted into a tendon which adheres to the lacrymal bone. The orbicularis is supplied by the seventh nerve, and when this nerve is paralyzed the lids refuse to close, a staring expression being given to the eye.

Diseases of the Lids.

Lagophthalmos, or incomplete closure of the palpebral fissure, is usually occasioned by peripheral palsy of the seventh nerve, resulting from intracranial causes; it may, however, result from narrowing of

the lids by injury or ulceration, or by the formation of cicatrices (*cicatricial ectropion*), and it may ensue when the eyeball is enlarged or pushed forward (*proptosis*). (Fig. 116.)

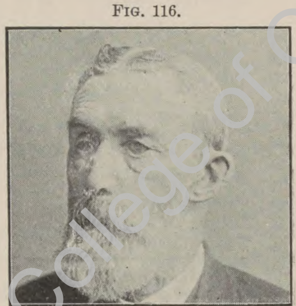
FIG. 115.



Position of Meibomian glands. (ARNOLD.)

When lagophthalmos is present as the result of an intracranial lesion, it may be accompanied by hemiplegia of the same or opposite side. Thus, a lesion anterior to the pons or in its anterior portion will cause palsy of the seventh nerve of the same side as the hemiplegia, whereas a lesion posterior to the pons or in its posterior portion will occasion palsy of the opposite side. On account of the exposure to the eye which lagophthalmos occasions, disease of the cornea and conjunctiva is frequently provoked. When the condition is due to facial palsy, active measures should be instituted at once for its relief; these include leeches and hot stupes, mercury and the iodides in specific cases; later the continuous current and hypodermic injections of strychnine. Until closure of the lid has been effected, the eye should be kept bandaged. If the cornea be threatened or if the condition becomes permanent, a tarsorrhaphy should be performed.

FIG. 116.



Lagophthalmos.

TARSORRHAPHY. The simplest way to shorten the palpebral aperture is to pare lightly the inner lips of the upper and lower lid-margins from the angle for 3 to 6 mm. or more, and then stitch them together without removing the eyelashes (de Wecker, Noyes). Tarsorrhaphy is usually done as follows: After gauging the length of the line of union required the lid is slightly everted and made tense—some stretch the canthus by inserting a spatula behind it. The margin of

each lid is split with a fine knife just behind the row of cilia and sufficiently deep to include the hair bulbs. The narrow flaps of skin with bulbs are then removed by incisions, meeting a little beyond the commissure. The inner lips are lightly pared from the latter to a point several millimetres beyond the end of the flap wounds. The raw surfaces are then carefully coapted by silk sutures passed obliquely up (or down) and in, and made to graze the inner lip. Traction is relieved by compresses and plaster for three or four days, when the sutures are removed. To make tarsorrhaphy more effective, Fuchs cuts away the flap with the hair bulbs from the upper lid only, splits the lower lid, and frees the anterior flap by a vertical cut at the inner end of the slit. The inner surface of the anterior flap is then stitched to the raw upper wound.

Tarsorrhaphy is indicated in the relaxed or everted lower lid of senile and paralytic cases, in lagophthalmos, in proptosis, and exophthalmic goitre; it is done often as an adjunct in blepharoplasty.

Blepharospasm, or spasm of the orbicularis, may be either *symptomatic* of other ocular disease, or *essential*. The latter variety is rare, and is often hysterical, while the former is a frequent accompaniment of many forms of ocular disease, especially where there is much dread of light, as in phlyctenular keratitis, and where a reflex irritation of the fibres of the trigeminus has been excited. In this connection should be mentioned the fibrillary contraction which occurs so frequently in a localized portion of the muscle. Apart from the annoyance which this occasions, it is not significant, and can usually be made to disappear by the adjustment of glasses to correct any existing error of refraction. Of similar origin are the attacks of "blinking" which occur in school-children. Not infrequently these are accompanied by choreic movements in the muscles of the face. In adults blepharospasm often is associated with tic. Blepharospasm is due not infrequently to hysteria, in which event "pressure points" may be found in the region supplied by the trigeminus, which will occasion opening of the lids when they are pressed upon.

Treatment. Treatment of blepharospasm will depend upon the cause. If the patient be anæmic, tonics should be administered; arsenic is of value in choreic cases; all errors of refraction should be carefully corrected. In cases arising from reflex irritation of the fifth nerve all possible foci of disease should be investigated and removed. Galvanism is useful in the later stages.

The lids are opened by the action of the levator palpebræ superioris and by the sinking of the lower lid by its own weight. The levator arises at the apex of the orbit and is inserted into the upper edge of the tarsus by three attachments. It is supplied by a twig from the third nerve, and when paralyzed the upper lid cannot be raised, and the condition called *ptosis* or drooping of the upper lid ensues.

Ptosis may vary in degree from a slight droop of the lid to the complete covering of the eyeball. It may be congenital, when it is

usually associated with epicanthus and affects both lids; usually, however, it is acquired. *Acquired ptosis* may result from palsy of the branch of the third nerve supplying the levator of the lid. It may, however, be due to local changes in the upper lid, which increase its volume or weight, such, for example, as occur in trachoma and various tumors of the lid. *Paralytic ptosis* may be due to peripheral or central cause, and is associated frequently with palsies of other branches of the third nerve. If isolated, it is due usually to an intracranial lesion.

Treatment. Antisyphilitic and antirheumatic treatment should be tried in suitable cases; galvanism and hypodermic injections of strychnine are of value in the later stages. For slight degrees of ptosis resulting from inefficiency of the levator, owing to injury or ancient trachoma, etc., the removal of a narrow horizontal strip of skin and muscle may suffice with insertion of the deeply placed sutures brought out high up on the lid. This operation is more certain if a piece of tarsal cartilage also is excised, as in the Gillet de Grandmont operation (Harlan), in which case the horizontal tarsal wound is united by buried catgut sutures. Advancement of the levator by the Eversbusch (which see) and the Snellen methods also gives good results.

Ptosis Atonica (Hotz). In this condition the skin of the upper lid hangs down over its free edge when the eye is open, and, instead of following the upward movement of the tarsal cartilage, remains stationary. It is due to loss of the normal connection between the skin and tarsus, and is corrected by the Hotz operation used in trichiasis and entropion.

Ptosis adiposa, in which a layer of fat beneath the skin acts mechanically and causes falling of the lid, if not overlapping, is relieved by removing the mass of adipose tissue through an incision in the skin.

To correct much drooping of the lid after trachoma, Gruening employs a modified Hotz suture. "An incision is made just below the upper edge of the tarsus and parallel to it. Some orbicularis fibres may be excised. With a sharply curved needle the thread is pushed along the surface and upper edge of the tarsus through the conjunctiva, until it loops up the cul-de-sac and returns upon itself beneath the skin, to come out at the upper part of the wound. It never traverses the skin. Three sutures may be used. They are tied tightly and allowed to remain two to five days, according to the amount of reaction" (Noyes).

Eversbusch's operation in partial ptosis has given excellent results. A horizontal incision is made across the lid and half-way between the lid-margin and the eyebrow; the tissues are then separated so as to expose a strip of connective tissue. A strong piece of catgut with a curved needle at either end is used. One needle is passed into the tendinous tissue as far as possible and brought out again a few millimetres from the point at which it was introduced. Both needles are

then passed parallel to each other and at 2 or 3 mm. distance apart below the skin and muscle of the lower portion of the lid, along the surface of the tarsus, and brought out at the free margin of the lid. Similar loops are next passed to the inner and outer sides of this central one. The clamp is then removed, the bleeding stopped, and the edges of the wound in the skin brought together with stitches.

Bowman first suggested the shortening and readjustment of the levator palpebræ (Snellen). In Wolff's operation, in which this is done, the tendon of the levator is exposed by a transverse incision. It is then undermined and two strabismus hooks are passed horizontally beneath it and apart as far as the difference in height of the two lids. Two double-needled catgut sutures are put into the tendon at the line of the upper hook, and it is then cut across just below the latter and is tucked behind the stump. The needles are then passed through the base of the stump and the sutures tied. The skin wound is stitched separately.

Mules' Operation for Ptosis. To secure the lifting action of the frontalis, Mules[1] inserts a permanent subcutaneous wire loop, "having its fixed points between the frontalis tendon and the lid cartilage. By this wire the lid is raised and maintained with great exactitude at a line whence a slight effort of the frontalis is sufficient to lift it to any required elevation. At the same time its folds are re-established and its normal appearance regained." By means of needles with eyes at their points the two ends of a fine wire (gilded iron or silver), which is inserted at a point in the tarsal plate near the ciliary border are brought out one-third of an inch above the brow and one-half of an inch apart. The ends are drawn up (the next day, A. S. Morton) until the lid is raised as desired, when one is run under the skin to the other, and they are then twisted and buried. Result very satisfactory (W. J. Cant, A. S. Morton).

Motais' Operation. Motais has designed an ingenious operation for raising the upper lid in congenital ptosis, by resecting a tongue-shaped flap from the centre of the tendon of the superior rectus and attaching it beneath the palpebral conjunctiva to the fascia, so as to act as a levator to the tarsus.

Pagenstecher's Operation. Pagenstecher's method of utilizing the frontalis in paralysis of the levator aims at securing a vertical subcutaneous cicatrix connecting the former with the lid. He passes a needle carrying a thick ligature under the skin of the forehead about one-half an inch above the centre of the eyebrow, and subcutaneously as far as the margin of the lid at its middle point. The suture is tied and tightened from day to day until it has cut its way out.

Gradle's modification is the insertion of permanent aseptic subcutaneous sutures.

Panas' Method. In this operation, which has pleased many operators, the skin of the brow and eyelid is steadied by pressure

[1] Ninth International Ophthalmological Congress, 1894.

across the forehead. Two horizontal incisions are made, the lower at the orbital margin, and along the top of the flap with a slight convexity upward, and not quite an inch long; the higher one a little longer, and at the upper border of the eyebrow. A flap of the skin and muscle is now dissected from the tarsus down to its ciliary border, but the septum orbitæ (suspensory ligament) of the lid is not disturbed. The bridge of tissue between the two horizontal incisions is undermined without cutting the periosteum or septum orbitæ. The flap is then drawn up under the bridge by means of sutures and fastened to the upper edge of the higher incision. When the flap is so fixed, the traction tends to cause ectropion, and a suture is therefore placed at each side, passing deeply through the septum orbitæ and conjunctiva, but not the skin, and it also is inserted in the upper lip of the higher incision, so as to correct the tendency to eversion.

Wilder's Operation. For the relief of complete ptosis W. H. Wilder has devised a method which "consists in folding upon itself the tarso-orbital fascia that connects the margin of the orbit to the tarsus and acts as a suspensory ligament for the upper lid." By shortening it with buried sutures the lid may be raised as desired, and in a number of cases the result has been satisfactory. The eyebrow is shaved, an incision one and one-half inches long is made parallel to the orbital margin and a little above it to the periosteum. The lower lip of the wound is drawn down and the skin and muscle are carefully dissected from the fascia, and the tarsus exposed. Sutures of fine sterilized catgut or silk armed at each end with a curved needle are passed in the following manner: The first needle is introduced sufficiently deep into the tarsus to secure a firm hold at a point about at the junction of the outer and middle third and a little distance from its convex. It is then drawn through, and with it several gathering stitches are taken in the tarso-orbital fascia, after which the needle is made to pass through the muscle and connective tissue of the upper lip of the wound. The other needle on the same suture follows a parallel course in the same manner, entering the tarsus about 3 mm. from the point of entrance of the first, then gathering the fascia into small folds and emerging in the tissue above, thus making a loop by which the lid may be drawn up. The second suture is passed in the same way, making a loop at the junction of the middle and inner third of the tarsus. The requisite elevation of the lid may be now secured by drawing on the loops and tying the sutures, which are to be buried in the wound. The lower lip of the wound is now united to the upper with fine sutures. The slight scar that remains after healing is almost entirely hidden when the eyebrows grow again. The buried sutures become encapsuled and give additional strength to the folds of fascia that hold up the lid. The orbicularis is uninjured, so that the patient retains to a certain extent the power of closing the lids.

The movements of the eyelids are both voluntary and involuntary. Winking is usually voluntary, but may be the result of a reflex action.

When the latter is the case, it is accomplished through the fibres of the fifth nerve, which supply the cornea and ocular conjunctiva, acting upon the orbicularis. The lids protect the eyeball from injury and excessive light. They aid also in lubricating the globe by distributing the tears over it and mechanically brushing away foreign substances which may have intruded themselves under the lids.

The arteries of the lids are derived chiefly from the ophthalmic artery; the veins are very plentiful, and empty partly into the veins of the forehead and partly into the ophthalmic vein.

Congenital Anomalies of the Lid. *Cryptophthalmos* refers to the stretching of the skin over the orbit covering the eyeball.

FIG. 117.

Coloboma of the lids.

Coloboma is a fissure in the lid, triangular in shape, with the base at the border of the lid, the apex pointing toward the margin of the orbit. It is a rare affection, and occurs usually in the upper lid. (Fig. 117.)

Epicanthus is the name given to crescentic folds of the skin which project on both sides of the face from the inner angle of the brow. Epicanthus is frequently associated with ptosis.

Treatment consists in excising an elliptical piece of skin from the root of the nose. Canthoplasty at the outer angles will often benefit the deformity.

CANTHOPLASTY. As a rule, the operation for the extension of the palpebral fissure and the weakening of the orbicularis is not a plastic one, and it is better styled canthotomy. Cocaine, 10 per cent. solution, is applied on a pledget within and without the external canthus, the spring speculum is inserted or the lids stretched apart, one blade of the scissors, which are held horizontally, is pushed behind the outer canthus toward the bony rim, and a quick snip suffices. The conjunctiva is freed slightly, and is then stitched at three points to the skin edge, at the angle, above, and below. If the orbicularis is hypertrophied and causing pressure on the globe, the external palpebral ligament is snipped, the scissors' points being passed into the wound vertically behind the muscle, the lid being drawn outward (C. R. Agnew). When there have been atrophy and shrinkage of the conjunctiva, it may be necessary after the section to stitch a piece of transplanted skin into the angle or adjust a small Thiersch shaving—canthoplasty.

CANTHOTOMY is a useful adjunct in some cases of blepharospasm from keratitis, to relieve pressure in purulent conjunctivitis and as a

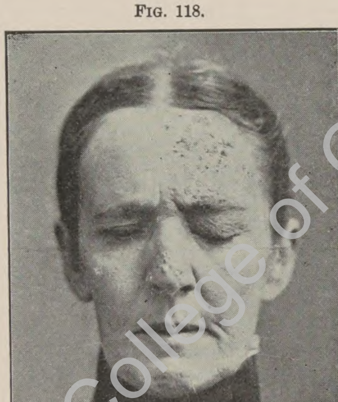
step in enucleation or exenteration. It is, as a rule, necessary in entropion, especially of the lower lid, and sutures are always used when a permanent effect is desired. The latter is the more sure if, as Jackson points out, a broad edging of the conjunctiva is taken up in the stitch.

Inflammation of the Lids. The integument covering the lids is liable to be affected by disease common to the skin in general, such as erysipelas, herpes, and eczema.

When the lids are involved in **erysipelas** secondary to a similar condition of the face, they may be so swollen that the ball is completely hidden, and at times the process is so active that the inflammation spreads into the deep tissues of the orbit, causing abscess and not infrequently blindness by involvement of the optic nerve in the orbit. Primary erysipelas of the lids is extremely rare. The treatment is that of erysipelas elsewhere in the body, both as to local applications and general medication.

Eczema of the lids also usually participates in a general eczematous eruption upon the face. It occurs commonly in children as a moist eczema (*crusta lactea*), when it is usually accompanied by a similar form of conjunctivitis. Disease of the lacrymal apparatus in adults not infrequently gives rise to eczema, particularly of the lower lid, by the irritation provoked by the overflowing tears. Treatment consists in the proper cleansing of the skin by alkaline washes, followed by the application of an ointment of oxide of zinc or of a solution of nitrate of silver (10 to 20 grains to the ounce). In the chronic form when there is much itching, carbolic acid may be added to the zinc ointment in the strength of 5 grains to the ounce.

FIG. 118.



Herpes zoster ophthalmos.

Herpes zoster (Fig. 118) not infrequently is the result of an inflammation which is situated either in the trunk of the fifth nerve itself or in the Gasserian or ciliary ganglia. A number of vesicles form along the terminal expansion of the trigeminus; at times these vesicles are limited to the distribution of the upper or the lower branch of the nerve; at times both of these branches are affected simultaneously, but it is rare that the inferior division is affected in common with the two superior. The efflorescences never extend beyond the median line of the face. For several days preceding the eruption there are severe pain in the course of the nerve and some febrile reaction, and the skin becomes red and swollen, resembling erysipelas. In severe cases an ulcer forms at the base of the vesicle, due to involvement of the corium in the process of suppuration, and a deep scar

forms; in milder cases the vesicles disappear without leaving any mark. Analogous affections of the cornea, conjunctiva, and iris may complicate the disease and give rise to cellulitis and opacities, which render the prognosis most unfavorable. The treatment is purely palliative, consisting in the application of a powder of rice starch or of an ointment of zinc, to aid in the drying up of the vesicle. The vesicle should never be opened. If the cornea becomes affected, appropriate remedies should be employed. Large doses of quinine and of salicylic acid are often of service. Galvanism may be used for the relief of persistent pain along the course of the nerve.

Abscess of the Lid. This is generally the result of injury, although it may be due to caries of the orbit, to periostitis, and to disease of the accessory sinuses. It may be a complication of erysipelas. In the first stages there are diffuse infiltration and redness of the lid. Later a localized swelling appears which points in some cases, but spreads in others, involving the whole lid in a gangrenous process. In the latter case extensive damage is wrought to the lid, and deformities result which may occasion lagophthalmos and ectropion.

Treatment If seen in the earlier stages, attempts should be made to abort the inflammation by means of ice-packs; if induration be present, free incision should be made with a view to checking further spread of the disease.

Furuncle, carbuncle, and **anthrax pustule** are rare. The two former present much the same symptoms as abscess, with the distinction of being accompanied by a small gangrenous slough or "core;" the latter is due to inoculation by the Bacillus anthracis, and occurs in persons who are occupied with the care of animals.

Ulcers of the Lid. These may be the result of local cause, such as injury or manifestations of a general disease—*i. e.*, syphilis, lupus, scrofula, herpes. The most frequent of the constitutional sores is the secondary ulcer of syphilis. This is usually found upon the skin near the margin of the lid or below the inner canthus; it occurs late, and might almost be regarded as a tertiary lesion.

Vaccina and **smallpox** not infrequently give rise to eruptions upon the lids. As the result of the cicatrization caused by these ulcers *madarosis*, or loss of the eyelashes, may occur, together with ectropion. A true vaccine ulcer (*vaccinia of the eyelid*) may be caused by infection from a vaccination sore. The border of the lid usually is affected, and considerable swelling and redness and involvement of the preauricular and submaxillary glands, with constitutional symptoms, accompany the ulcerous process. In the early stages the pustules are characteristic, though later they may resemble a syphilitic sore.

Œdema of the lid may be a symptom of a neighboring local disease, such as disease of the lids themselves or of the conjunctiva, or orbit, or it may be a manifestation of systemic disorder, such as disease of the heart or kidneys. It may be an accompaniment of an active inflammation of neighboring parts, or it may be due to simple venous congestion.

Recurrent necrotic œdema of the lids is associated frequently with similar swellings elsewhere, and is to be imputed to a temporary disturbance in the vascular innervation.

Syphilis of the Lid. In addition to the ulcers mentioned above, the lid may be the seat of a primary sore. An ulcer in this position, with a hard, indurated base, appearing without the history of injury, but followed by secondary manifestations, should always excite suspicion of syphilis. Soft chancres also occur upon the lid. *Tarsitis syphilitica* is a tertiary manifestation of syphilis affecting the cartilage of the lid. One or both lids may be affected. The lid becomes swollen and tense and the skin reddened; the cilia drop out. In favorable cases the swelling gradually subsides, leaving the lid in its original condition; in others, however, the tarsus becomes much distorted, and entropion results.

Blepharitis. (Fig. 119.) On account of the presence of the cilia and the hair follicles with their glands upon the margins of the lids, this portion of the lid is not infrequently the seat of inflammation.

Hyperæmia of the margin of the lid usually attends all forms of conjunctivitis; it is a frequent index of eyestrain, and may be occasioned in certain individuals by slight cause, such as dust, smoke, or foul air. The most common variety of inflammation of the lids, *blepharitis*, consists in a chronic condition which is associated with the formation of scales and crusts at the base of the cilia. Blepharitis occurs under two forms: *the superficial* or *non-ulcerative*, and *the deep* or *ulcerative*. In the first variety the margins of the lids are red and swollen and are covered with numerous whitish scales. If these are washed away, a few cilia drop out, but some grow in again.

FIG. 119.

Blepharitis. (DALRYMPLE.)

In the second variety the hair follicles become destroyed by ulcers which form about the roots of the cilia, so that the lashes fall out. This may occasion permanent loss or displacement of the cilia, hypertrophy of the margin of the lid, and ectropion. In the milder cases of blepharitis the patients suffer but slight discomfort, but when the inflammation has been of long standing, increased lacrymation, sensitiveness to light, itching, and burning render the patient very miserable. The causes of blepharitis may be general or local. Among the former may be mentioned the exanthemata, especially measles, a debilitated system, and unhygienic surroundings; uncorrected errors of refraction are a frequent cause. The local conditions

which may occasion it especially are disorders in the lacrymal apparatus and conjunctiva. Blepharitis is frequently hereditary, and is more common in children than in adults.

Treatment consists in the removal of the cause. The correction of errors of refraction and attention to the systemic condition should be insisted upon, and in many cases a cure will be accomplished without resorting to other measures. If the blepharitis be due to lacrymal or conjunctival disease, these should be combated by proper local treatment. Before making any applications to the edge of the lids it is first necessary to remove all scales and crusts adherent to them. This may be accomplished by washing the lids thoroughly with soap and water or with water containing borax. In the superficial variety of blepharitis a salve of mercury (yellow or red oxide, gr. viij, vaseline ℥j; or the ammoniated chloride of mercury in the same strength) should be applied; in the ulcerative variety an application of a solution of nitrate of silver (1 to 3 per cent.) to the raw spots on the lid is often of service. This should be followed by an application of mercurial ointment smeared thickly upon lint and lightly bandaged upon the eyes over night. If abscess occur, the cilia should be removed by means of proper forceps (*epilation*).

Phthiriasis Ciliorum (Blepharitis Pediculosa). This is an affection of the margin of the lid due to the presence of the Pediculus pubis in the lashes. It is frequently mistaken for blepharitis, and usually occurs in children. Treatment consists in cleansing the lids with a solution of mercuric chloride, 1 : 4000, and subsequently rubbing one of the mercurial ointments into the border of the lids.

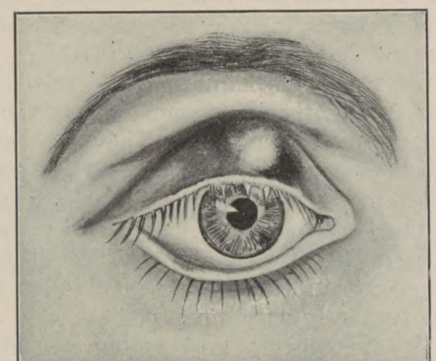
Hordeolum (Stye). This is a hard circumscribed swelling on the lid margin, a grain of barley in size, which generally suppurates. The inflammation occurs in the tissues about a hair follicle, the Meibomian glands not being involved. Owing to the tension which is created by the pus between the tarsus and the skin, there is usually considerable pain until the contents of the stye have been evacuated. As a rule, the process lasts four or five days. Repeated attacks are common. Hordeolum occurs usually in the young, especially in those who are anæmic and debilitated. Errors of refraction may induce the condition, as well as exposure to local irritation, such as heat and dust. Blepharitis is a not infrequent cause.

Treatment. Ice-packs may be used as an abortive, but as soon as swelling appears hot applications are to be employed to favor suppuration. Evacuation of the contents should be practised by incision as soon as a yellow spot forms. All refractive errors should carefully be corrected and the general health cared for. Calcium sulphide, one-eighth grain three times daily, is of service in recurrent cases.

Chalazion (Fig. 120) is a chronic disease of one of the Meibomian glands as a consequence of the stoppage of its duct, and results in the formation of a small tumor in the lid. The growth of the tumor is slow, with moderate or no signs of inflammation until at the end

of a few weeks or months it has attained the size of a large pea. Chalazia adhere to the tarsus, but the skin is mobile over them, and they are not usually sensitive to the touch. They may become absorbed and disappear spontaneously; but, as a rule, they break down, suppurate, and discharge their contents either through a skin or conjunctival opening. Chalazia are found in adults particularly. They rarely occasion pain, but are disfiguring and may cause symptoms of eyestrain by the pressure which they exert upon the eyeball.

FIG. 120.

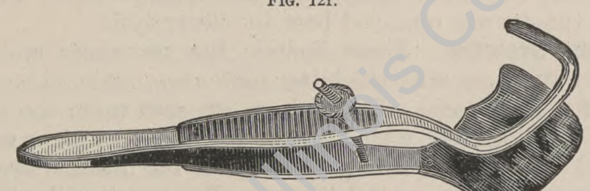


Chalazion.

Treatment. Unless giving rise to irritation, small chalazia need not be interfered with; large chalazia should be removed by incision through either the skin or conjunctiva.

Chalazion is removed, as a rule, through the conjunctiva. The lid is everted and the free edge pressed well back, cocaine hydrochloride applied to the site, and a drop or two of 10 per cent. solution of cocaine injected hypodermically. A short vertical cut is made from within out, and the tumor, if small, is then emptied with a fine

FIG. 121.



Desmarres' chalazion forceps.

serrated or sharp-edged scoop. When large and with thick wall, the latter is grasped with fine fixation forceps and cut out with a sharp-curved scissors. If only in part, the cavity is scraped to remove acini, and the contents packed at the sides. Bleeding is often free, when adrenalin chloride, 1:5000, applied early and also pressed into

the cavity, is of use. Hemorrhage may also be controlled and the field of operation rendered bloodless by applying a Desmarres chalazion forceps. (Fig. 121.) If there are several chalazia in a bunch, a lid clamp or ring forceps is used. If the chalazion is large and hard, or shows signs of pointing externally, it may be removed through the skin by a horizontal incision with use of clamp. Fine stitches are then inserted. Iced compresses for a few hours are soothing. If the chalazion is near the free edge, it may be opened with a fine cataract knife through the lid margin, the lid being clamped between the index finger-tip in the cul-de-sac and the thumb (C. R. Agnew).

Tumors. *Benign growths* include xanthelasma, molluscum, cysts, warts, and cutaneous horns, and vascular tumors or angiomata. *Xanthelasma* is a flat yellowish plaque slightly raised above the skin, which occurs most frequently in women and at the inner canthus. These plaques are often symmetrical. They are caused by degeneration of the muscle fibres. Their growth is slow, and as they occasion no bad results other than disfigurement, they need be removed only for cosmetic effect. *Molluscum* is a small white growth which forms on the lid as a result of a diseased condition of the sebaceous glands. It occurs in two forms: *molluscum contagiosum*, in which variety the tumor is without a pedicle, and has an umbilicated depression in its centre; and *molluscum simplex*. In the latter variety the tumor is pediculated, hanging from the lid like a pouch.

Cysts. Among these may be mentioned dermoid cysts, milia, and antharomata.

Angiomata. These comprise *telangiectasis* and *tumores cavernosi*. The former occur as small bright-red growths in the skin of the lid, and are due to dilatation of the bloodvessels. The latter are distended venous channels beneath the skin. Both varieties are usually congenital and occur after birth. Care should be exercised in their removal, to avoid cicatrices. Small telangiectases may be removed by the thermocautery or by cauterization with nitric acid; large ones should be seared to foster contraction and obliteration of the vessels. Cavernous tumors are removed best by electrolysis.

Malignant Growths. These include the *sarcomata* and *carcinomata*. The former are rare, the latter more common, and occur under the form of *rodent ulcers*. These ulcers are seen upon the margin of the lid as a small pimple, which breaks down into an ulcer with indurated walls. These ulcers slowly spread over the lids and occasionally dip down deep into the orbital tissues. Treatment of both forms of tumor consists in their early and complete removal by surgical intervention.

Blepharoplasty. To meet the loss of lid-tissue from disease and injury, or its necessary sacrifice in removing neoplasms, etc., new material has, of course, to be provided. Unless the gap in the lid is such as permits closure by stretching what is left, new material has to be secured, either from adjoining parts—the forehead, temple,

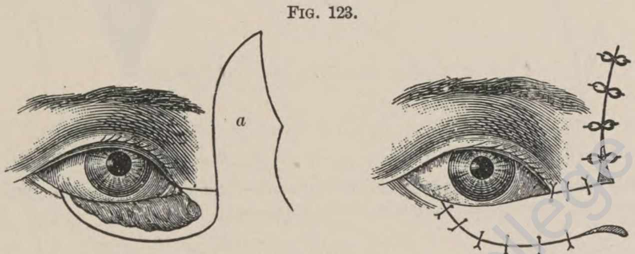
cheek or nose—by means of flaps with pedicles or from other regions by flaps without pedicles or by skin-grafts.

Flaps with twisted pedicles are often used after Fricke's method, in which the base abuts one end of the raw surface. The gap left by the flap may be covered by Thiersch or Wolfe grafts, or, if not too large, by undermining the limiting skin and suturing the edges together. In the employment of sliding flaps by Dieffenbach's method, which has been much practised, a more or less vertical and quadrangular flap, at the side of the gap—which is made fairly triangular—is slid into place and stitched. Its bed is covered by Thiersch or Wolfe grafts either at once or after a day or two, or later by smaller dermic grafts. Knapp's method[1] of stretching horizontal flaps (Fig. 126) is a distinct addition to blepharoplasty. By it one may

FIG. 122.

Arlt's method of removing a growth from the canthus. (Arlt.)

FIG. 123.



Fricke's method of blepharoplasty. (Arlt.)

remove a neoplasm requiring the sacrifice of most, if not all, of the lower lid, and then cut a flap, going beyond the bridge of the nose, and a longer one with broad base on the temple, and unite them in vertical line, so as to restore a useful eyelid, tarsorrhaphy being also done. The writer, following the suggestion of C. S. Bull, has found systematic massage of cicatricial keloid and other scar-tissue a valuable adjunct to blepharoplasty.

The Le Fort-Wolfe transplantation of flap without pedicle and the Thiersch skin-grafting have proved a great gain to blepharoplasty and a boon to operators, who need not now run the risk that they mar more than they mend. Wolfe's method was designed specially for

[1] Archives of Ophthalmology, vol. xiii.

cases in which skin is needed to replace cicatricial tissue, or the latter surrounds the damaged lids and is insufficient to supply flaps. Thiersch skin-grafts are sometimes more suitable. In the performance of the Wolfe operation, which is especially well adapted to cases where, as a result of burns or injury, the free edge of one lid has become attached to the brow or the cheek, the lid is freed by careful dissection.

The lid is freed by careful dissection until both lids can be easily approximated, when they are sutured at three or four apposed points where the lid margins have been pared. The raw surfaces are trimmed until they are smooth and vascular. "Having taken the shape of the flap required with a piece of lint, I carefully dissect a piece of skin from the forearm, cutting it larger than necessary all around, so as to allow for shrinking. I then turn up its deep surface, and with a pair of flat, sharp scissors pare off every vestige of subcutaneous tissue, so as to leave the surface of a pure white color. It

FIG. 124.

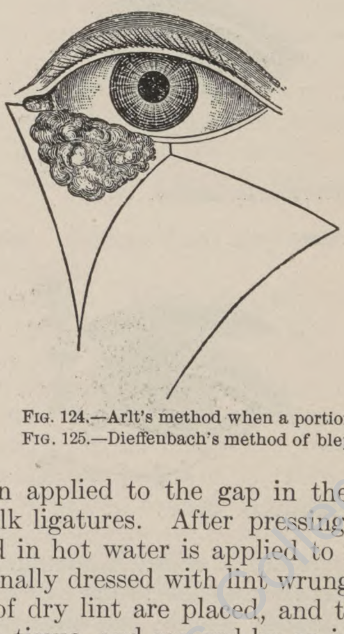


FIG. 125.

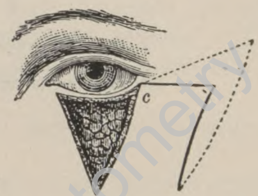


FIG. 124.—Arlt's method when a portion of the eyelid is to be sacrificed. (ARLT.)
FIG. 125.—Dieffenbach's method of blepharoplasty. (ARLT.)

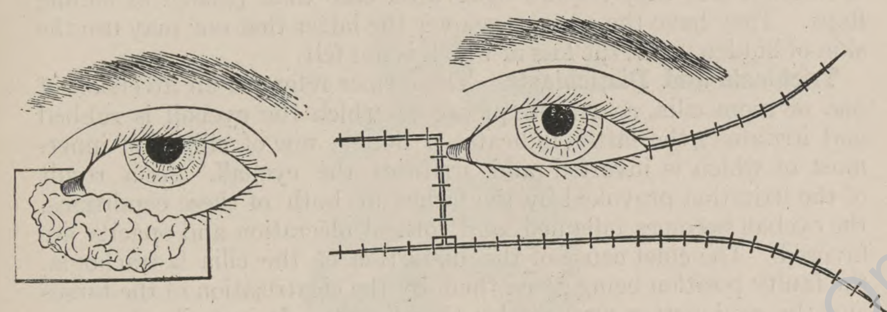
is then applied to the gap in the eyelid, to which it is united by fine silk ligatures. After pressing and moulding it into place, lint soaked in hot water is applied to its surface for about ten minutes. It is finally dressed with lint wrung out of hot water. Over this four folds of dry lint are placed, and the whole covered with fine gutta-percha tissue, and secured by an immovable bandage. On the third day the dressing is removed, and it is found that adhesion had taken place, the flap looking clean and dry and normal; bluish lines are visible here and there. The same dressing is repeated daily."

In Thiersch's method of skin-grafting, strips of epidermis and superficial layers of the skin, cut generally from the hairless part of the arm, are transferred to the raw surface prepared as in Wolfe's method, the lids being stitched together or the upper to the cheek. While making the shaving, the parts are kept wet with warm physiological salt solution, and the former is slid off the razor and across

the raw surface by its aid, all clots having first been removed. Dressing is done as in the Wolfe method. It is well to bandage both eyes. In forty-eight or seventy-two hours the parts are carefully exposed and redressed, and then from time to time. The ligatures can be removed in a week. More or less shrinking of the flap, 25 to 30 per cent., may occur.

In cicatricial ectropion following burns, etc., in which at least a portion of the eyebrow has been destroyed, Hotz[1] has obviated the necessity of the Wolfe method and the danger of re-eversion of the lid from shrinkage of a transplanted flap or retraction of tissues. From the adjacent cicatricial skin itself he takes "a flap large enough to cover the lid surface only, and fixes the upper margin of this flap to the upper border of the tarsus." The raw surface above the lids is covered by another flap or Thiersch grafts. The incision begins

FIG. 126.



Knapp's method of blepharoplasty. (KNAPP.)

near the inner canthus and ends about 6 mm. from the outer, and skirts a large skin flap, which is then carefully dissected off from the underlying scar-tissue, but is left connected with the lid border. Then the lid is released from the deeper scar tissue until it can be replaced in its normal position. The contracted flap, still, however, large enough to cover the whole surface of the lid, is spread out smoothly over this surface, and its margin is fixed to the upper border of the tarsus by four silk sutures. Into the wound upon the lid a skin flap, which may also contain a good deal of scar-tissue, is transplanted.

In blepharoplasty—apart from asepsis and great care—there are some points that conduce to success. The flap should be a third larger than the gap, should have a broad, thick base, with as good vascular supply as possible, and be neatly adjusted with the least twisting, and the least strain upon the sutures. The latter should not be put in until all bleeding has ceased and clots are removed. The thread should be fine and of twisted (not braided) silk, because leaving less mark, and the stitches, which should be closely placed,

[1] Archives of Ophthalmology, vol. xxv., No. 3.

should be removed early. The flap for the lower lid should be taken, when it is feasible, from a higher level to prevent sagging or eversion from after-traction, which is partly met by tarsorrhaphy. For the upper lid the flap is often taken from the mid-frontal region. After restoration of the lids the parts should be kept warm and quiet, and free from special tension, by means of compresses, plaster, and bandage, so adjusted as to avoid undue direct pressure. The natural folds and creases of the adnexa should be kept in mind. Tissue, especially conjunctiva, should not be wasted, though neoplasms should have wide berth. Hence, in this class the importance of early correct diagnosis and prompt excision.

Flaps with pedicles have not been discarded because of the Wolfe and Thiersch methods, for these have their drawbacks. Flaps without pedicle shrink from 33 to 50 per cent., and sometimes more, and the original defect may recur. Not seldom they slough in part or wholly, and they require more after-care than twisted or sliding flaps. They have the advantage over the latter that one may use the skin of hidden parts, the loss of which is not felt.

Trichiasis and Distichiasis. The former refers to an inversion of one or more cilia, as a consequence of which the eyeball is rubbed and irritated; the latter indicates a double row of cilia, the innermost of which is inverted and irritates the eyeball. As a result of the irritation provoked by the lashes in both of these conditions, the eyeball becomes inflamed, and corneal ulceration and opacity are favored. The chief cause of the distortion of the cilia is trachoma, the faulty position being given them by the cicatrization of the tarsus and the conjunctiva wrought by this disease. It may also be occasioned by injuries and blepharitis. Treatment consists either in the removal of the cilia or operative measures to correct their faulty position.

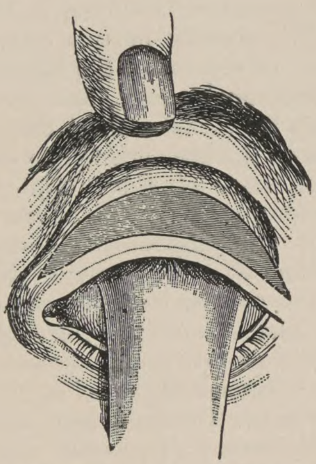
Trichiasis. When malposition is limited to only a few of the cilia, it may be corrected by excising an oval piece of the tissues near the free edge into the tarsus and stitching the skin wound (Wolfe); also by splitting the edge of the lids behind the row and putting in a tiny piece of skin or mucous membrane. In so-called scalping, the lid-margin is split vertically behind the stunted and incurved lashes, and the anterior lip, just wide enough to hold the hair bulbs, is abscised. This is now very rarely done, being replaced by the Burow incision, Green or van Millingen operation, etc. (which see). Electrolysis is now used to destroy misplaced cilia when they are few. A fine needle (negative pole) is passed into the follicle with the cilium as guide. A few seconds closure of the circuit suffices if there is frothing (Mitchell, Benson).

Spencer Watson's Method. An incision is made in the intermarginal space, and a second one parallel to the border of the lids and above the row of cilia, as is done for their ablation. This strip, containing the cilia and follicles, is cut across at one end only. Then a second flap similar in shape is made above the first, its free end

being at the same canthus as the base of the other; the flaps are then interchanged and sutured. This operation is now rarely done, except for trichiasis near one or other canthus. In these positions Fuchs considers it the most suitable procedure, but the flaps are made much shorter than in the original operation, which was, indeed, the pioneer in intermarginal work.

THE JAESCHE-ARLT OPERATION for trichiasis, which is done under an anæsthetic, is as follows: A Snellen or Knapp lid clamp is applied, and the lid-margin is split from end to end by an incision two lines in depth, which is met at the bottom by a horizontal cut through the skin at right angles, made about 4 mm. above the ciliary border. The anterior flap, holding the skin, orbicularis, cilia, and bulbs, is then cut away. A small semilunar piece of skin is now dissected

FIG. 127.



Jaesche-Arlt operation on the upper eyelid. (ARLT.)

off higher up, and the marginal flap is then stitched to the upper raw edge. The effect is to roll out the edge of the lid and the eyelashes. To make the result more lasting, Waldhauer trims the excised skin and fits it in the wound, and supports by bandage to ensure union with the raw surface. This operation has largely been replaced by reconstruction or restoration of the lid-margin (after van Millingen, Green, Hotz).

Entropion and **ectropion** refer to an inversion and eversion of the margin of the lid, respectively. *Entropion* may be occasioned by a defect in the normal contour of the lid as the result of a disease or injury of the conjunctiva or tarsus (*cicatricial entropion*), or it may be caused by a spasm of the orbicularis muscle acting reflexly from a conjunctivitis or keratitis, or from bandaging of the eyes, especially in the aged, with lax skin and conjunctiva (*spasmodic entropion*).

On account of the irritation which the inturning of the lashes upon the globe provokes, lacrymation, photophobia, and the signs of conjunctivitis and keratitis are the rule.

Treatment consists in restoring the margin of the lid to its proper position. If the entropion is due merely to spasm, it may often be relieved by painting the skin below the lid with collodion. It may be avoided in bandaging by applying a strip of adhesive plaster over the lids.

Operations for Entropion. When spasmodic or muscular entropion tends to persist in spite of the use of plaster or collodion, etc., some operation is required. Excision of a horizontal strip of skin often suffices in senile cases, or of a narrow strip of skin and muscle down to the tarsus close to the free edge of the lid (Green). Many cases have been cured by means of deep vertical sutures which transfix the septum orbitæ near the infra-orbital margin, essentially on the principle of the Hotz operation (Gruening). The Gaillard-Arlt suture is also effective. Four threads in two sets, by means of double threaded needles, are entered under the skin of the (lower) lid close to its edge and at right angles to it, and are brought out at 2 cm. straight below. Each set forms a short loop outside the skin near the cilia, and traction on the free ends when tying (over a quill) everts as desired. The sutures are left a few days, so as to cause vertical subcutaneous cicatrices which ensure permanent tension.

In entropion with narrowing of the fissure (blepharophimosis), a good result may be had by canthotomy combined with the insertion of deep vertical sutures passing from the ciliary margin close to the outer surface of the tarsus and emerging high up in the lid. The firmly tied ligatures are allowed to suppurate out; in some cases they are taken out early (Pagenstecher). For the correction of senile entropion of the lower lid, Theobald uses caustic potash, after the manner of the late Professor N. R. Smith. The crayon is sharpened to a point, and is moved back and forth across the lid at about 4 mm. from its margin. A spreading of the caustic action of perhaps 2 mm. from the line of application is to be allowed for by simply causing the potash to act upon this narrow strip of tissue parallel to the lid-margin, and moving it back and forth gently, perhaps a dozen times, one secures a very marked caustic action upon the tissue, which may be checked if desired by diluted acetic acid. As a rule, the lid will immediately stay out in good position; a slough takes place, and the cases in which a complete cure is not effected in senile entropion are very unusual. The method is not appropriate for the upper lid.

Trichiasis and Cicatricial Entropion. To correct the incurving of the lid-margin, bevelling of the inner lip and malposition of the cilia, caused by shrinking of the conjunctiva and the constant traction inward in the last stage of trachoma, three methods may be adopted: that of counter-tension, release from tension, and restoration (reconstruction) of the lid-margin. The first is the principle of the Anagnostakis and Hotz operation, which has stood the test of years. In

it counter-tension is kept up by using as a fixed point the tarso-orbital fascia at the orbital margin of the tarsus. The second and third objects are gained by the Green operation, very widely used, which frees the incurved lid edge and restores its inner lip, and also by the van Millingen operation, and in a different way, namely, by interposing a barrier between the skin of the lid and its conjunctiva.

J. Green's Operation for Cicatricial Entropion. A longitudinal incision is made through the conjunctiva and tarsus, from one end of the tarsus to the other (after Burow), and parallel to and about one line or one and a quarter line distant from the free border of the lid. A strip of skin, a little more than a line in width, and about a line distant from the row of eyelashes, is excised, the loosened margin of the lid turned forward and secured in its new position by from three to five sutures. The needle carrying the suture is made to enter at the edge of the lid, in or near the row of cilia, and is carried upward just beneath the skin until it appears in the cutaneous wound. It is then plunged deeply through and behind the fibres of the orbicularis muscle, and is brought out through the skin from a third to a half of an inch above the upper lip of the wound. The effect of the row of sutures applied in this way is to tilt forward the margin of the lid with the implanted cilia, leaving the longitudinal wound on the conjunctival surface to heal by granulation. When the tarsal cartilage has been very much thickened, a wedge-like strip from the upper portion is removed before excising the strip of skin. Care is taken to spare the underlying fibres of the orbicularis muscle when removing the narrow strip of skin. And where no skin can be spared or needs excision—the majority of cases—Green, after the deep tarsal incision, paints a strip along the entire length of the lid with contractile collodion. The lid is thus everted without loss of skin or use of sutures. "The gaping incision in the tarsus fills rapidly by granulation, and is soon covered by smooth conjunctiva. The increase in the height of the tarsus by this formation of new tissue is generally not less than two millimetres." A fine strip of mucous membrane may be inserted with good effect in the sulcus made by the Burow-Green tarsal incision.

The Green method is best adapted to the upper lid; the Green-Ewing is intended for the lower, and differs only in the use of the quill suture and in covering a portion of the incision with mucous membrane. The conjunctiva from the outer canthus to the punctum is dissected back from the free edge three to four millimetres, within which line the usual deep tarsal incision is made the full length of the lid. Three sutures are then put in the ciliary tarsal strip at the incised part, and then through the skin, and are tied over a firm roll of aseptic gauze or absorbent cotton about 3 mm. in diameter. Then the conjunctival flap is brought forward by numerous fine sutures and stitched deeply into the groove so as to cover with an epithelial surface both the raw edge of the divided tarsus and the exposed fibres of the orbicularis.

Anagnostakis and Hotz Operation. The operation is performed on the upper lid as follows: While an assistant fixes the skin at the supra-orbital margin, the operator, seizing the centre of the lid-border with fingers or forceps, draws the lid downward to put its skin well on a stretch, and makes a transverse incision through skin and orbicularis muscle from a point 2 or 3 mm. above the punctum lacrymale to a point 2 or 3 mm. above the external canthus. This incision divides the lid-skin in a line parallel to and a little below the upper border of the tarsal cartilage, and is therefore from 4 to 8 mm. distant from the free border in the centre of the lid. The skin and muscular layer are now dissected from the incision down to the roots of the eyelashes, and, while an assistant is holding the edges of the wound well separated the operator seizes the forceps and excises with curved scissors the muscular fibres running transversely across the upper border of the tarsus. Next the sutures are inserted. Three sutures are usually sufficient—one in the centre of the wound and one at each side of the central suture. The curved needle, armed with black silk, No. 3, is first passed through the wound-border of the lid-skin, then it is thrust through the upper border of the tarsus and returned through the tarso-orbital fascia just above this border, and finally it is carried through the upper wound-border. When these sutures are tied the skin is drawn upward and fixed to the upper tarsal border, and this slight traction is sufficient to turn the inverted lid-border and eyelashes to their normal position, and as the skin becomes firmly united with the tarsal border the tension thus produced upon the lid-border is permanently secured. The sutures should, of course, not be tied until all bleeding has ceased and the wound is thoroughly cleansed; they may be removed on the third day. Under aseptic dressings the wound heals by first union, even if, as sometimes occurs, secondary hemorrhage or œdema causes considerable swelling for several days.

The van Millingen Operation for Trichiasis.[1] "The intermarginal space is split from end to end, as in Arlt's operation, and sufficiently to produce a gap 3 mm. in breadth at the central part of the lid, and gradually becoming narrower toward the canthii. The gap is kept open by sutures passed through folds of skin on the upper lid and by means of which the lid is prevented from closing for twenty-four hours at least. As soon as the bleeding has ceased, a strip of mucous membrane of the same length as that of the lid, and 2 or 2½ mm. in breadth, is cut out with two or three clips of a pair of curved scissors, from the inner surface of the under lid, and placed at once into the gap at the intermarginal space. It should then be pressed into situ with a pledget of cotton-wool steeped in sublimate solution. Sutures are superfluous, and do more harm than good. The operated lid is then covered over with a flap of linen containing a thick layer of iodoform vaseline, and this is covered over

[1] Ophthalmic Review, November, 1887, vol. vi., No. 73.

by cotton-wool. Both eyes should be bandaged. I invariably use sublimate lotion (1: 5000) for disinfecting the eye and lid during, before, and after the operation. The bandage should be renewed once in twenty-four hours, and the sutures on the upper lid should not be removed before the second day." Some prefer a strip of skin (from behind the ear, Hotz), without suturing. Others with sutures. Some, like van Millingen, are partial to mucous membrane (from the lip, Weeks), using sutures or not.

THE STREATFEILD OPERATION of grooving the tarsus when it is thick and misshapen has been modified by Snellen. In the Streatfeild-Snellen operation an incision is made through the skin of the upper lid about 3 mm. from the margin and parallel to it and extending along its whole length. A strip of the orbicularis about 2 mm. in width is excised, and next a triangular wedge-shaped piece of the tarsus along the whole length of the lid. Three sutures are then inserted in the following manner: A suture armed at each end with a needle is to be passed through the upper edge of the incision in the tarsus, and both needles are then to be carried through the lower margin of this groove and brought out through the skin just above the line of lashes, the points of exit lying 4 mm. apart. The two other sutures are to be inserted in the same way, care being taken that the points of exit are about 4 mm. from each other. A bead is then passed over each end of the sutures (to prevent their cutting the skin), and the latter carefully tied, so that the two opposite sides of the incision in the tarsus are accurately approximated. The upper edge of the skin wound is left open.

PANAS-SNELLEN OPERATION. The skin of the lid is divided 2 or 3 mm. above the free border of the lid and parallel to the latter, the incision running the whole length of the lid. Then from the edge of this incision the skin is freed as far up as the upper border of the tarsus and as far down as the free border of the lid. Next, the vulcanite plate being inserted beneath the lid, an incision is made which runs in the same way as in the skin. In this way the lower half of the tarsus with the free border of the lid is made freely movable, and may be rotated forward by means of sutures, so that the cilia assume the proper direction. The sutures are formed into loops and passed above through the edge of the tarsus and the tarso-orbital fascia, and the free ends of the loop are brought out behind the skin of the lid along the intermarginal line, and here are tied over a glass bead.

The Green and the Hotz operations yield, as a rule, very satisfactory results; also the van Millingen, especially in trichiasis of the lower lid, combined with canthoplasty and the Streatfeild-Snellen when indicated. In cases of misshapen tarsus—and they are not a few—it may be necessary to combine the features of two, if not three of these, to ensure success, and this is the rule with operators. Weeks reports favorably of a number of cases in which he tried a combination of the four—canthoplasty, the van Millingen, the Streatfeild-Snellen, and Hotz—the suturing differing from that of the latter in that the

needle is made to pass through the upper lid and to emerge 8 mm. above the margin of the upper flap. The writer, who can testify to the value of the Hotz and Green operations, early found it advisable (before the van Millingen operation was in vogue) to combine the first at times with the Burow tarsal incision.

Like entropion, *ectropion* may either be cicatricial or spasmodic. (Fig. 128.) *Cicatricial*, or, as it is sometimes called, *organic ectropion*, results from wounds, abscess of the lid and orbit, and disease of long standing of the lids and conjunctiva. Senile relaxation of the skin may occasion ectropion of the lower lid in elderly subjects, and a similar condition also arises after paralysis of the seventh nerve. *Spasmodic ectropion* is seen usually in children when the lids are everted by violent blepharospasm produced by keratitis. The consequences of ectropion are irritation of the globe, due to the lack of protection afforded by the lid, and troublesome watering of the eye.

FIG. 128.

Double cicatricial ectropion.

FIG. 129.

Arlt's operation for ectropion. (ARLT.)

Treatment. The Adams operation was devised to correct elongation and partial eversion and moderate ectropion. It consists in the removal of a triangular V-shaped piece, including the whole thickness of the lid, the base of the triangle being turned toward the margin of the latter and the apex toward the cheek. The edges of the wound are carefully brought together by sutures, one of which is passed close to the free edge, so as to prevent a groove. Sometimes a harelip pin and suture is used. This operation is now preferably done near the outer canthus in conjunction with tarsorrhaphy. It is specially adapted for senile ectropion or that due to chronic conjunctivitis, for which also the Snellen and the Argyll-Robertson suture operations have been designed.

In the Argyll-Robertson operation[1] two needles threaded on a long waxed silk ligature are passed through the skin and lid one line from its ciliary margin, and each one-quarter of an inch from the mesial, passed on through the fornix and brought out through the skin one-quarter of an inch apart at one to one and one-quarter inches from the ciliary border. A bunch of fine rubber tubing is placed vertically

[1] Edinburgh Clinical and Pathological Journal, December, 1883.

within the loop on the outside of the lid. A piece of thin sheet-lead one by one-quarter inch, rounded off and moulded, is slipped into the cul-de-sac under the threads, and the ligature is then tied over the lower end of the tubing. "The edge of the lid is thus made to revolve inward over the upper edge of the piece of lead, while the tarsal cartilage is caused to mould itself to the curve of the lead, and the eyelid at once occupies its normal position." The sutures are not removed for from five to ten days.

In the Snellen-suture operation for senile or muscular ectropion the ligatures are passed through the conjunctiva and subadjacent tissues, and brought out and tied over a roll 2 cm. below the free edge. The effect may be increased by tarsorrhaphy as desired.

In partial ectropion with hypertrophy of conjunctiva, the excision of a horizontal strip of the latter and closure by sutures may suffice, or again the contraction following a deep linear eschar made with the fine thermo- or galvanocautery point.

One of the best operations for the cure of cicatricial ectropion is that of Arlt, which is performed as follows: The cicatrix and the skin surrounding it are excised in a triangular area bounded by the points *a b* and *b c*, as shown in Fig. 129. The skin at the edges of the denuded area is undermined and sutures inserted so that *c* is approximate to *d*, and the side of the flap *b c* is in contact with *c d*. Harelip pins may be used to fill in any remaining gap.

When considerable tissue is removed it becomes necessary to fill in the gap by a flap. This may be accomplished by either the Fricke or the Dieffenbach method. Both of these consist in excision of the cicatrix and the sliding of skin flaps into the denuded area. These flaps are taken from the skin of the temple or cheek, and vary in shape and size with the site which they are to occupy. Account should always be taken in all flap operations of the contraction which occurs both at the time of and subsequent to the operation. It is advisable, therefore, to make the flap at least one-third larger than the site which it is intended to occupy. Its base also should be sufficiently broad not to interfere with its blood supply.

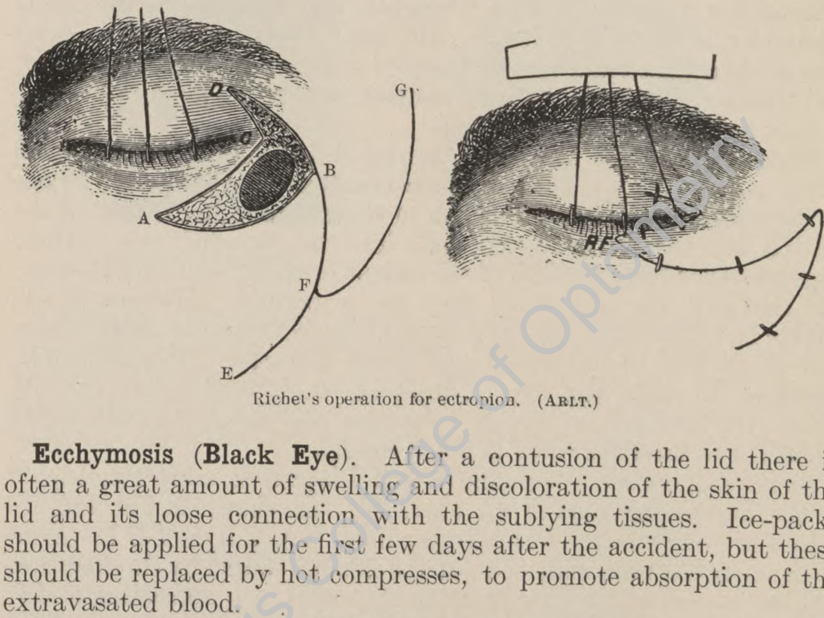
Richet's operation is peculiarly well adapted to correct ectropion of the lower outer part of the lower lid. As shown in Fig. 130, incisions are made in three curvilinear lines, B A, C A, and D E. After the cicatrix has been dissected out the lids are united by three sutures and the wound covered by drawing the edges into apposition as follows: An incision, F G, is made into the skin of the temple, and its edges undermined. The flap D F G thus obtained is made to cover in the defect, F being brought to A, as shown in the second figure. The denuded area which is left in the temple is filled in by the lower flap, A B E.

Ankyloblepharon and Symblepharon. Ankyloblepharon is an adhesion of the margins of the lids, and is usually associated with a union between the lid and eyeball (*symblepharon*). Both conditions result when the borders of the lid and conjunctiva are converted

into raw surfaces, either from burns or diseases of the conjunctiva, causing loss of tissue, such as trachoma and diphtheritic conjunctivitis.

Treatment. Ankyloblepharon is remedied by dividing the adhesions between the lids and between the lids and the globe, and by covering the denuded areas by transplanted portions of conjunctiva or strips of mucous membrane taken from the lips. In many cases it is of advantage to perform a canthoplasty at the same time. The steps in the operation for the cure of symblepharon consist in separating the adhesions between the lid and the globe, and in preventing readhesions between the denuded areas by placing a conjunctival surface in apposition with a raw one. A number of procedures are available, but that of Himly or Harlan is to be preferred. The former perforated the base of the attachment of the adhesion in the cul-de-sac and placed a strip of lead wire in that position, the wire being permitted to remain until it had worn a groove covered with epithelium.

FIG. 130.



Richet's operation for ectropion. (ARLT.)

Ecchymosis (Black Eye). After a contusion of the lid there is often a great amount of swelling and discoloration of the skin of the lid and its loose connection with the sublying tissues. Ice-packs should be applied for the first few days after the accident, but these should be replaced by hot compresses, to promote absorption of the extravasated blood.

Emphysema indicates that there has been a fracture of the walls of the orbit establishing a connection with the nose. As the air is forced into the lid by blowing the nose, the patient should be cautioned against this act until the wound is healed, and a firm compress bandage should be applied over the eye.

Injuries of the Eyelids. These may be a mere incident of a more serious traumatism, and hence, if feasible, the features of the accident should be learned. Wounds should be explored for possible foreign bodies in the eye, orbit, and adnexa, and one will sometimes be sur-

prised at the findings. Foreign particles should be removed, and wounds be made aseptic at the earliest possible moment and closed with fine twisted silk. The lips of wounds dividing the edges of the lids should be carefully coapted to avoid distortion or a groove. A fine suture in the lid-margin itself is often useful. If the levator palpebræ is torn, it should be stitched with fine catgut. When skin is torn away the gap should be closed by sliding flaps or be covered without delay by Thiersch grafts. Burns which destroy much skin of the lid or act deeply necessitate the stitching of the lids together (see Tarsorrhaphy), and probably the use of Thiersch grafts shortly, to prevent secondary ectropion. Aseptic wounds of the lids heal rapidly and without reaction, but in some cases the ice-bag or iced dressings are helpful.

CHAPTER VI.

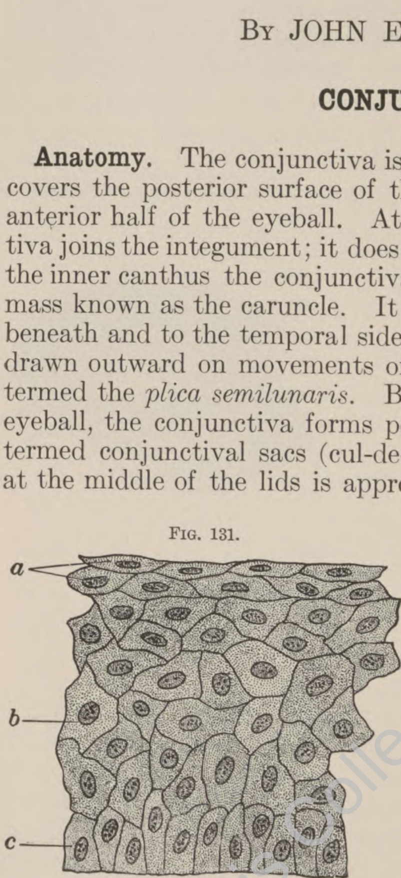
DISEASES OF THE CONJUNCTIVA, CORNEA, AND SCLERA.

By JOHN E. WEEKS, M.D.

CONJUNCTIVA.

Anatomy. The conjunctiva is a delicate mucous membrane which covers the posterior surface of the eyelids and is reflected onto the anterior half of the eyeball. At the margin of the lids the conjunctiva joins the integument; it does not pass beyond either canthus. At the inner canthus the conjunctiva extends over the fleshy glandular mass known as the caruncle. It is thrown into a crescentic fold just beneath and to the temporal side of the caruncle. This fold, which is drawn outward on movements of the cornea to the temporal side, is termed the *plica semilunaris*. By its reflection from the lids to the eyeball, the conjunctiva forms pouches above and below, which are termed conjunctival sacs (cul-de-sac). The depth of the upper sac at the middle of the lids is approximately 19 mm., of the lower sac 8 mm. The conjunctiva is divided into various parts, as follows: *palpebral*, that covering the posterior portion of the lids; *fornix*, the transition fold; *ocular*, that covering the globe. The part of the palpebral portion that covers the tarsus is known as the *tarsal conjunctiva*. At about three millimetres from the margin of the cornea the conjunctiva becomes closely united with the anterior reflection of Tenon's capsule. The epithelial layer is stratified. (Fig. 131.) The tunica propria is very thin. The conjunctiva possesses no large vessels, but it has a very rich network of small vessels, which become prominent on irritation of the conjunctiva. In the retrotarsal and ocular portions of the conjunctiva the vessels are freely movable over the underlying tissue.

FIG. 131.



Epithelioma of the bulbar conjunctiva. (ORTH.)

A rich plexus of lymphatic vessels exists in the conjunctiva, those of the upper conjunctiva near the outer canthus being in connection with the chain of lymphatic vessels which pass to the preauricular

region; those of the lower lid are connected more directly with the submaxillary lymphatics.

The nerve supply is from the lacrymal and from the supratrochlear and infratrochlear branches of the fifth.

Congenital Abnormalities. The most frequent congenital growths met with are dermoid tumors, which usually extend onto the cornea; they are usually pale in color, but may be pigmented; they are, as a rule, supplied with a number of hairs, glands, etc., presenting the characteristics of the skin. Small fatty masses are also met with; these are situated apparently beneath the conjunctiva.

Angioma, cavernoma, and telangiectatic growths, congenital in origin, sometimes are found in the conjunctiva.

An osseous growth occasionally is found beneath the conjunctiva, situated between the outer margin of the cornea and the commissure.

Moles or pigmented patches sometimes are observed on the conjunctiva; these occur most frequently in individuals who present similar spots on the skin.

Fibrous growths, sometimes erroneously spoken of as lipomata, occur rarely just beneath the membrane in the upper outer portion of the ocular conjunctiva.

Hyperæmia of the Conjunctiva (Dry Catarrh). The palpebral conjunctiva is the part usually affected. The mucous membrane is red and very slightly roughened, but is not appreciably thickened.

Etiology. The condition is due to irritation from many causes—exposure to heat, bright light, glare from water, sand, etc., strong wind, cold, storms of rain or snow, constant use of the eyes with insufficient illumination, eyestrain, indigestion, alcoholism, gout, vasomotor disturbances, lacrymal disease, acute exanthematous fevers, and blepharitis marginalis.

Symptoms. The lids feel stiff and dry, and are moved with difficulty; a burning sensation is experienced, and there is increased lacrymation. The superficial epithelial cells are thrown off more rapidly than in health, and are found in small whitish masses at the canthi and sometimes at the margin of the lids. Attempts to use the eyes with artificial light are accompanied by distress.

Treatment. The cause should be sought for and removed. In addition the eyes should be bathed twice daily with a 3 per cent. solution of boric acid. Other measures are unnecessary.

Conjunctivitis (Ophthalmia). This term is applied to a number of diseases of the conjunctiva, all of which are accompanied by increased and altered secretion, by distressing symptoms, and by transient or permanent pathological changes in the membrane.

Classification. Since the discovery of the gonococcus of Neisser, in 1879, the specific micro-organisms of a number of forms of conjunctivitis have been described, which makes it advisable to modify the older classification of diseases of this membrane. All of the forms of conjunctivitis may be included under two headings: (1) those in which a specific cause has not been determined, and (2) those forms in

which a specific cause has been determined. To the first class belong: simple conjunctivitis, lacrymal conjunctivitis, herpetic conjunctivitis, vernal conjunctivitis, follicular conjunctivitis, trachoma, pemphigus, Parinaud's conjunctivitis, gouty conjunctivitis. To the second class belong: acute contagious conjunctivitis, subacute conjunctivitis, gonorrhœal conjunctivitis, diphtheritic conjunctivitis, xerosis epithelialis, phlyctenular or eczematous conjunctivitis, tuberculosis, lupus, leprosy.

Non-specific Forms of Conjunctivitis.

Simple Conjunctivitis (Catarrhal Conjunctivitis, or Ophthalmia). This condition is characterized by injection and slight thickening of the conjunctiva confined almost entirely to the palpebral portion, loss of transparency, slight roughening, and the presence of a very little mucus, which causes the lids to adhere together in the morning.

Causes. These are numerous and permit of classification:

1. Mechanical. Irritation of the conjunctiva, due to the entrance of particles of metal, dust, pollen, exposure to wind, glare of light.

2. Associated. Accompanying the exanthematous fevers, rheumatism, nasal catarrh, bronchitis, eczema, facial erysipelas, impetigo contagiosa, molluscum contagiosum.

3. Symptomatic. Forms accompanying eyestrain, etc.

Symptoms. Subjective. Lids heavy, burning sensations in the eye, irritation on moving the eyes, photophobia, annoyance in use of the eyes.

Objective. Lacrymation more profuse, slight sticking together of the lids in the morning, slight thickening of the lids, hyperæmia of the tarsal conjunctiva and of the retrotarsal folds.

Simple conjunctivitis, as is apparent by a glance at the list of causes, is most common in children, but no stage of life is exempt.

Prognosis. The duration depends on the continuation of the cause; when this is removed (mechanical and symptomatic forms) or subsides (associated forms), recovery occurs spontaneously. No lasting injury results.

Treatment. In addition to removing the cause, much relief may be obtained by bathing the eye two to four times daily with a solution of boric acid, 3 per cent. A mild astringent solution may also be employed—zinc (gr. j to ℥j) is excellent.

Lacrymal Conjunctivitis. A form of conjunctivitis dependent on the presence of irritating secretion from the conducting portion of the lacrymal apparatus. Almost all of the cases might properly be classed with the simple conjunctivitides, but a few cases develop a purulent type which may result in much damage to the eye.

Diagnosis. The symptoms peculiar to obstruction of the lacrymal passages and, frequently, a dacryocystitis are present.

Treatment. This consists in rendering the lacrymal canals patulous and in correcting the condition of the lacrymal conducting apparatus;

these measures may be supplemented by cleansing the conjunctiva with some bland aseptic solution and the use of a mild astringent.

Lithiasis Conjunctivitis. A form of irritation of the conjunctiva due to the presence of calcareous deposits in the tissue of the palpebral conjunctiva; they occur most commonly in the tarsal conjunctiva, but are met with also in the palpebral portion of the retrotarsal folds. The small masses appear as yellowish-white bodies almost immediately beneath the epithelium. They tend to penetrate the epithelial layer and to produce irritation of the eyeball and lids. The condition is met with commonly in those of advanced years, and is associated usually with rheumatism or gout. The deposits consist principally of calcium carbonate and cholesterin.

Treatment. Removal.

Herpes Conjunctivæ. This condition is characterized by the formation of clusters of vesicles on a hyperæmic base. The vesicles collapse, forming a superficial ulcer which heals rapidly, leaving a very slight superficial cicatrix. The affection accompanies herpes orbitalis, and will be described under that head.

Vernal Conjunctivitis (**Conjunctivitis Catarrhalis Æstiva; Phlyctena Pallida** (Hirschberg); **Spring Catarrh**). A disease characterized by roughening and thickening of the palpebral conjunctiva, accompanied by hypertrophy of the conjunctiva at the margin of the cornea.

Cause. While this disease is in all probability due to a specific germ, the germ is not known. The exacerbations occur when the weather becomes warm, whatever the season; but the peculiar appearance of the tarsal conjunctiva is not entirely absent in the winter months. Children from the age of three to fifteen years are attacked most frequently; but the condition sometimes appears in adults. Often two or more in a family are attacked, a fact which points to a contagious quality. In almost all cases both eyes are affected.

Symptoms. Irritation, as of a foreign body, photophobia, distress on use of eyes, burning and itching; there is excessive lacrymation; a scanty mucoid (stringy) discharge, which is evidenced in the morning by a yellowish-white mass along the lashes and at the inner canthus. On everting the upper lid, the tarsal conjunctiva is found to be slightly thickened and the surface is roughened by the presence of numerous fine, papilliform elevations. The surface of the palpebral conjunctiva both above and below presents a faint, pearly hue, as though a drop of skimmed milk had been passed over it. This appearance is observed in the early as well as in the later stages of the disease. The ocular conjunctiva, except at the margin of the cornea, is but slightly affected; at the limbus the epithelial layer becomes much thickened. This thickening is usually greatest in the horizontal meridian. The elevations have a pearly, translucent appearance at the apices which is characteristic. The hypertrophied tissue often encroaches onto the cornea for a distance of one or two millimetres, and a narrow grayish zone separates the hypertrophied tissue from clear cornea.

In the later stages in severe cases flattened fungoid elevations appear on the palpebral conjunctiva of the upper and lower lids. These often resemble trachoma granules. They may be pedunculated.

Pathology. The changes in the conjunctiva consist in scanty small-cell infiltration and the development of papillæ, particularly on the upper tarsal conjunctiva. These papillæ consist of a central loop of vessels and some connective-tissue stroma covered with a layer of thickened stratified epithelium. It is undoubtedly the thickened epithelium that gives the whitish shimmer to the surface. In severe cases fungoid excrescences form, consisting of a fibrous papilla covered by thickened stratified epithelium.

Prognosis. The disease recurs every summer for a variable period (two to twenty years), when it subsides, usually leaving but little deformity.

Treatment. Protective glasses, a bland wash (boric acid solution), and the use of an ointment of the yellow oxide of mercury (1 to $1\frac{1}{2}$ per cent.), usually give the best results so far as remedies are concerned. Calomel, in impalpable powder, dusted onto the palpebral conjunctiva in very thin layer every second day is advantageous. Climatic changes do most good; the sufferer should go to a cool climate during the hot months.

Follicular conjunctivitis (**conjunctivitis folliculosis simplex**) is characterized by the appearance of small, pinkish, translucent oval elevations arranged often in rows which occupy the outer portion of the fornix of the lower lid, occasionally being present at the outer and inner portions of the palpebral conjunctiva of the upper lid.

Cause. There is no known specific cause, but the disease occurs most frequently in children who live in unhygienic surroundings, and the evidence is in favor of filth as a cause. The disease is infectious, perhaps contagious.

Symptoms. There is often considerable irritation; the lids are slightly thickened. There is some mucoid secretion on the lids in the morning. Use of the eyes causes sensations of burning and smarting. The ocular conjunctiva and cornea are seldom involved. On inspection, the palpebral conjunctiva is found to be congested, and the follicles are prominent.

Treatment. The eyes should be bathed three or four times daily with a 3 per cent. solution of boric acid, and a solution of mercuric chloride, 1:1000 to 1:4000, should be dropped into the conjunctival sac after each bathing. Aristol, iodoform, bismuth, and calomel, equal parts, or calomel alone may be employed. In persistent cases expression of the contents of the follicles may be resorted to. Errors of refraction should be corrected. To prevent spreading of the disease, isolation should be resorted to, especially when it occurs in asylums.

Trachoma (**Granular Conjunctivitis; Egyptian Ophthalmia; Military Ophthalmia**). This disease is characterized by the presence of

numerous small oval masses in the palpebral conjunctiva, by chronicity, and by grave subsequent changes in the conjunctiva, lids, and often in the globe. It occurs most frequently in children, but may affect individuals at any age except perhaps during the first year of life.

Description. Trachoma may be conveniently divided into three stages:

First, the stage of *hypertrophy*, in which the granules are discrete, and the area of the conjunctiva is as great or greater than in the normal, no cicatricial tissue having formed. This stage presents three distinct phases:

(*a*) The granules develop without discomfort to the patient, very little mucous secretion being present—not sufficient to seal the lids in the morning; there is a slight excess of lacrymation, and the lids appear slightly thickened. There is no redness of the ocular conjunctiva, nor is the cornea affected.

(*b*) This is the form of onset most frequently observed. The patient complains of pain in the eyelids, which feel hot and rough. There is evidence of pronounced irritation, lacrymation is increased, and in a few days after the discomfort is first experienced a mucopurulent discharge is present, not however in large quantity; the lacrymation continues, the ocular conjunctiva becomes injected, and even in the relatively early part of this stage the cornea may give evidence of involvement. On everting the lids, the conjunctiva is found to be deeply injected and thickened; and if the inflamed condition has lasted two to four weeks, granules may be seen on the tarsal conjunctiva and possibly in the retrotarsal folds. Often the hypertrophy of the conjunctiva is sufficient to mask the presence of the granules, and they become visible only after the swelling of the conjunctiva has subsided. The preauricular glands are enlarged. When occurring in residential schools, asylums, reformatories, and in families, the disease spreads rapidly, and, unless isolation is practised, many of the inmates become affected.

FIG. 132.

Conjunctiva of upper lid in chronic granular conjunctivitis. (ARLT.)

(*c*) This phase is fortunately rare; it is the most severe, usually affecting young and middle-aged adults. The onset is rapid. Burning and scratching of the lids are complained of. The lids become moderately swollen. There is lacrymation, and in a day or two a mucopurulent and sanguinolent discharge. Hypertrophy of the conjunctiva is present after a few days; at the end of ten days or two weeks the conjunctiva is greatly thickened, the entire fornix presenting a plaque of lymphoid tissue. The ocular conjunctiva becomes deeply injected, and it is not unusual to observe evidence of corneal irritation early in the course

of the disease. The preauricular lymph glands are swollen, and in some cases the submaxillary glands are similarly affected.

The first stage of trachoma may last six weeks to a year; it gradually passes into *the second stage,* which may be termed *the stage of coalescence* or *beginning of cicatrization.* This stage is common to the three phases of onset, appearing later in the first phase than in either of the others. The granules, which before were discrete in the first two phases of onset, coalesce, and cicatricial tissue appears in the form of narrow bands throughout portions of the palpebral conjunctiva. The area of the conjunctival surface diminishes, and the cul-de-sacs decrease in depth; with this change the tarsus becomes narrower and shorter and abnormally acutely curved. The rough surface of the lids rubs against the cornea and destroys its epithelium. Vascular pannus forms, superficial ulcerations of the cornea follow, and if pathogenic germs find entrance to the corneal tissue, deep ulcers, with more or less destruction of the cornea, ensue. The margins of the lids become inverted (entropion), and the lashes rub against the cornea. The palpebral fissure is narrowed.

Trachomatous tissue may appear on the ocular conjunctiva, the caruncle, or even on the cornea. Years may elapse before the second stage passes into the *third stage,* which is known as *the stage of atrophy* or *cicatrization.* The cornea now presents an opaque appearance. The conjunctiva is much reduced in area, and presents none of the appearances of the normal mucous membrane; the surfaces are dry, except perhaps for the presence of a few islets of approximately normal tissue. Vision is reduced to perception of light; the conjunctival surface as well as the cornea is dry (xerosis cicatricialis) and pale in color.

Trachoma need not necessarily pass through all of these stages, but may be arrested, with the preservation of what normal tissue remains at any part of the first or second stage. The disease ceases spontaneously in rare cases, but too frequently persists throughout the life of the patient if treatment is not resorted to.

Causes. While trachoma is not confined to the poor, it is much more frequently met with among them, filth, overcrowding, vitiated atmosphere, and improper and insufficient food contributing to its production. It is possible that a contagium must be added to produce the disease. Many researches have been undertaken to discover the specific cause, and a micro-organism has been isolated which bears a close relation to the disease; this micro-organism, which is a small double coccus, has been described by Sattler and Michel. Mutlermilch has described a fungus which he terms Microsporosa trachomatorum. Pfeifer and Ridley have described parasitic protozoa. Although it is believed to be a microphytic disease, sufficient evidence is not yet at hand to establish the identity of any known germ as the specific cause.

It cannot be demonstrated that any condition of the system predisposes to trachoma. It is found in the robust as well as in the

poorly nourished. Lymphatic individuals do not appear to contract the disease more readily than others.

Pathology. The trachoma follicle, which is substantially a miniature lymph gland, is the essential element; these follicles consist of a delicate indefinite connective-tissue capsule containing a mass of lymphoid cells, this collection of cells being traversed by very fine connective-tissue trabeculæ. (Plate V.) Small bloodvessels ramify in the connective-tissue stroma that surrounds the follicle, and capillaries are found in the mass of cells that form the follicle. As the disease passes into the second stage, the septa between individual follicles disappear and the lymphoid masses become continuous, forming plaques of various sizes, and the substantia propria of the conjunctiva gradually gives place to cicatricial tissue. The epithelium covering the granules varies in thickness and is irregular.

Diagnosis. Trachoma in its first stage may be confounded with vernal catarrh, tuberculosis of the conjunctiva, and Parinaud's disease. The history of the case will suffice to distinguish it from the first, or, if the history is not sufficient, microscopical examination of a nodule will suffice. In vernal catarrh the nodule is a fibroma. The microscopical examination with the history of the case will suffice to distinguish it from tuberculosis, and in Parinaud's disease the excessive involvement of the cervical and preauricular glands with the affection confined to one side (as it usually occurs) will be sufficient.

Prognosis. This is favorable when the disease is seen in the first or early part of the second stage. When the cornea has become involved, further damage may be obviated; but the tissues destroyed cannot be restored.

Treatment. This is prophylactic, medicinal, and surgical. Trachoma should be treated as a contagious disease. In homes care should be taken to require the patient to sleep alone and to prevent other members of the family from using towels, handkerchiefs, washing utensils, etc., that are employed by the patient. In asylums, barracks, etc., isolation with individual towels should be enforced.

MEDICINAL. The eyes should be thoroughly cleansed as often as is necessary to keep them free from discharge, by bathing with a solution of boric acid or mercuric chloride (1 : 15,000); eyedrops of mercuric chloride (1 : 5000 to 1 : 3000), formalin (1 : 3000), chlorine water (50 per cent., U. S. P.), or chloride of zinc (gr. j to ℥j), may be instilled into the eye three or four times daily; the conjunctival surface may be sprayed once daily with tannic acid and glycerin (gr. xxx to lx to ℥j). Boroglyceride (30 to 50 per cent.) may be applied to the surface of the conjunctiva, and is of value in the later stage, when there is more or less xerosis. Iodide of benzosinal, 2 per cent. (Seabrook), may be of service. Jequirity bean in infusion and in powder is employed to excite a counter-inflammation to cause absorption of the follicles.

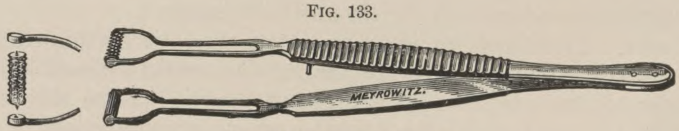
Solid Applications. The remedy that finds most favor in cases of trachoma where the discharge is not profuse is the crystal of the

sulphate of copper; this is applied by lightly and rapidly passing the smooth crystal over the affected portions of the conjunctiva. The sulphate of aluminium and potassium crystal and the stick of mitigated nitrate of silver are also employed with benefit.

Of the powders, iodoform, iodol, aristol, boric acid, or calomel may be dusted on the affected parts after cleansing. Corneal complications usually require atropine, but nothing additional.

Surgical. Comprises the various methods employed for removing the granules. Galezowski, in 1874, advocated removal of the retrotarsal fold under ether anæsthesia by seizing the fold with forceps, drawing it down and excising it. The operation should never be resorted to, as it is too destructive of conjunctival tissue.

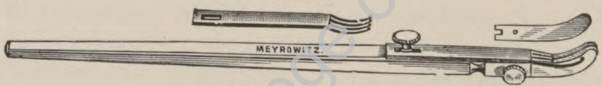
Fig. 133.



Knapp's expression forceps.

The method that has found most favor is that of expression, for which a number of instruments have been devised, known as expression forceps. (Fig. 133.) In 1889 Prince presented a "ring" forceps for this purpose before the Illinois State Medical Society. Other forceps are those of H. D. Noyes, Knapp, and Gruening. Scarification of the epithelial covering of the granules before expression is attempted facilitates escape of the contents. For this step, the Desmarres scarificator or that of the writer may be employed. Fig. 134.) As the operation is painful, ether anæsthesia is desirable, but cocaine may be used. The lids are fully everted, the surface

Fig. 134.



Weeks' scarificator.

superficially scarified, and the shallow incisions directed parallel to the margin of the lid. The folds of conjunctiva are seized with the forceps and freed from the trachomatous tissue by a gentle stripping motion. Afterward the surface may be bathed with a weak sublimate, boric acid, or normal saline solution, and treated by cold compresses without bandaging, or a bandage may be applied for twenty-four hours; the introduction of an ointment of mercuric chloride in vaseline (1 : 5000) serves to prevent adhesions between the conjunctival surfaces and to exert a mild antiseptic effect. The after-treatment consists in the use of a mild antiseptic wash three times daily and in breaking up any adhesions that may form. Cocaine anæsthesia should be employed when

these adhesions are attacked. In cases where the palpebral fissure is narrowed and the cornea is suffering from undue pressure from the lids, canthoplasty may be resorted to.

Parinaud's conjunctivitis is a mucopurulent affection of the conjunctiva characterized by the formation of rather large granules or elevations on the conjunctiva, which sometimes are pediculated (Gifford). The condition is accompanied by pronounced swelling of the preauricular, retromaxillary, and cervical glands, which sometimes suppurate.

The onset of Parinaud's disease, so far as the eye is concerned, is much like that of acute trachoma. Lacrymation is followed in forty-eight to seventy-two hours by a mucopurulent secretion, with swelling of the lids, which in severe cases is pronounced. On everting the upper lid on the third or fourth day, elevations are observed which resemble the granules in acute trachoma; these nodules generally become somewhat larger than in trachoma, and soon superficial ulcers are observed in the sulci between the small nodules. The ulcers seem to bear some relation to the degree of involvement of the preauricular and cervical glands; when the ulcers are numerous the glands are most severely affected. Ulceration of the cornea, which is an occasional complication, is also more apt to occur when the conjunctival ulceration is most marked. Chills and fever accompany the affection.

Cause. The disease is supposed to be due to an infection of animal origin; it attacks individuals of all ages, is monolateral, and does not appear to be contagious. No specific micro-organism has been discovered, although the nature of the disease points strongly to a specific cause.

Duration. The disease may terminate in three weeks, or it may persist for six or eight months. Relapses are very apt to occur, but eventually perfect recovery takes place.

Treatment. No special treatment directed to the nodules is necessary. Frequent cleansing with a solution of boric acid seems to be sufficient. A solution of mercuric chloride (1: 3000) may be instilled every four hours, or calomel may be dusted onto the lids

Gout of the Conjunctiva. An intense œdematous swelling of the conjunctiva of the lids and of the eyeball, accompanied by profuse lacrymation, with little mucus, and occasioning great discomfort to the patient, is sometimes met with in individuals who suffer from gout. This form of conjunctival irritation closely resembles the manifestations of gout as observed in the swelling of the great toe, the œdema of the ankles and other distal articulations. It appears suddenly, reaching its height in twenty-four to forty-eight hours, and recedes in five to ten days. Chemosis may be marked. It is usually accompanied by gouty manifestations in other parts of the system, and is the occasional manifestation of a gouty crisis.

Treatment. Locally, cleansing of the conjunctival sac three or four times daily with a solution of boric acid and the application of

cool lead-and-opium wash compresses to the lids. Internally, treatment should be directed against the gouty condition.

Pemphigus. This affection, which runs a specific course, but for which no cause has as yet been determined, is extremely rare, occurring but three times in 70,000 cases of eye disease observed by Horner. It is characterized by the formation of transient bullæ, which form on the palpebral and at times on the ocular conjunctiva, on a reddened base. The bullæ soon break, leaving a floor slightly paler than the surrounding conjunctiva, with shreds of epithelium hanging to its borders; the denuded surface is soon covered by new-formed epithelium, and the process is shortly repeated. The denuded surfaces become agglutinated to opposing denuded surfaces, and soon meridianal bands of connective tissue join the ocular and palpebral conjunctiva. Cicatricial tissue slowly forms in the substantia propria of the conjunctiva, and after many years the conjunctival sac becomes obliterated and superficial ulceration of the cornea develops. A condition of cicatricial xerosis is gradually reached and vision is reduced to perception of light.

Cause. Pemphigus usually accompanies pemphigus vulgaris or pemphigus foliaceus, and depends on a dyscrasia of the system. It attacks individuals at all ages.

Treatment. Treatment is of little value.

Forms of Conjunctivitis in which the Etiological Factor has been Determined.

Acute contagious conjunctivitis (pink eye) is due to the presence of a small bacillus known as the Weeks and as the Koch-Weeks bacillus (Plate VI., Fig. 1), first mentioned by Koch in 1883, and proved to be the specific micro-organism by Weeks in November, 1886.

Susceptibility. All conjunctivæ are susceptible to the influence of this micro-organism. One attack of the disease does not produce immunity.

Symptoms. For thirty-six to forty-eight hours after the inception of the contagium nothing further than a slight itching of the eye is experienced. On the morning of the second day the margins of the lids are stuck together by a mucopurulent secretion. There is a burning sensation in the lids. Interference with vision is slight. Toward evening the mucopurulent secretion increases and the general discomfort is more marked. By the fourth day the secretion has assumed a yellowish color and is quite copious. The height of the disease is usually reached on the third or fourth day. The acute stage lasts from three to seven days, and may be accompanied by coryza and frontal headache. In the early part of the acute stage numerous small extravasations of blood are frequently observed in the ocular conjunctiva. This symptom is so pronounced that some English surgeons have termed the disease "hemorrhagic conjunctivi-

tis." The congestion of the ocular conjunctiva in the acute stage gives the eye a vivid red appearance, which has caused this form of conjunctivitis to be popularly known as "pink eye." As the acute stage subsides, the secretion becomes less copious but thicker. A bright-yellow mass of secretion is present at the inner canthus in the morning, a sign that is almost pathognomonic of the disease. With subsidence of the secretion and of the swelling of the lids and conjunctiva, the painful symptoms disappear; however, a sensation of dryness of the conjunctiva persists for weeks, particularly noticeable on use of the eyes with artificial light.

Duration. If simple cleanliness is observed, the disease usually runs its course in two or three weeks, all of the symptoms disappearing. It may last for six months if no treatment is instituted. Under suitable treatment the average duration is eight to twelve days.

Contagious Qualities. Mucopurulent conjunctivitis is extremely contagious. In residential schools, asylums, barracks, penal institutions, communities, and families, it frequently becomes epidemic. It may become endemic. Communication from one individual to another is, probably, by means of towels, common bathing water, etc., as well as by direct contact. There is little doubt that the contagious element may be carried by draughts of air and by the water in public baths.

Diagnosis. In a typical case the diagnosis is comparatively easy. In severe cases the condition may be mistaken for gonorrhœal conjunctivitis, or even for diphtheria in cases where a pseudomembrane occurs. The microscope is necessary to clear up the diagnosis in these cases.

Complications. Phlyctenulæ, pseudomembrane, corneal ulcer (rarely).

Prognosis. Favorable in all cases.

Prophylaxis is all-important, and consists in strict quarantine until all of the secretion has disappeared.

Treatment. Cold applications to the lids for one hour at a time three times daily during the acute stage. Frequent cleansing with sterile boric acid solution (3 per cent.) should be resorted to. As the acute stage begins to subside much benefit is derived from applying a 0.5 per cent. solution of nitrate of silver to the conjunctival surface once daily until the secretion ceases.

Pneumococcic Conjunctivitis. Acute contagious conjunctivitis, due to the presence of the pneumococcus first described as a cause of conjunctivitis by Morax, in 1894. The clinical features of this disease are similar to those of the affection just described, except that, as a rule, the disturbance is less severe. The description and treatment just given will suffice for this form of conjunctivitis.

Contagious Qualities. Pneumococcus conjunctivitis affects children and adults, and it may become epidemic. It has been proved by the researches of Gasperini, Gifford, and others, that a susceptible condition of the conjunctiva must exist before the disease in question

can be produced. It is well known that the Weichselbaum pneumococcus may exist in the normal conjunctival sac without producing inflammation.

Diagnosis. It is difficult to differentiate it from conjunctivitis due to the small bacillus. The microscope will serve to establish the diagnosis.

Duration. Time, three days to two weeks.

Prognosis. Good in all cases.

Subacute Conjunctivitis (Diplobacillus Conjunctivitis). This form of conjunctivitis is insidious in its onset, producing redness and slight thickening of the conjunctiva, largely confined to the conjunctiva of the lids and fornices. There are slight increase in lacrymation, a scanty secretion of mucus, with some pus corpuscles, irritation as of a foreign body in the eye, and burning sensations on use of the eyes. The annoyance is relatively slight, but persistent. The eyelids may become somewhat congested, but they are not appreciably thickened. In rare cases the cornea becomes involved, a superficial marginal keratitis being produced, followed by cloudiness of the affected area. This may advance and narrow the transparent area of the cornea to very small limits.

Cause. Morax, and later Axenfeld, have described a bacillus as the cause of this disease, and their studies have been confirmed by Gifford and others. The bacillus measures 2 to 3 μ in length, and 1 to 1.5 μ in breadth. (Plate VI., Fig. 2.)

Duration. The disease may last for six weeks or as many months.

Contagiousness. Very slight.

Treatment. The eye should be cleansed with a boric acid solution, and zinc chloride (gr. j to ℥j) should be instilled twice or three times a day.

Gonorrhœal Conjunctivitis (Purulent Conjunctivitis; Acute Blennorrhœa). This disease is described under two titles, namely, gonorrhœal ophthalmia and ophthalmia neonatorum, the latter term being applied to the disease as it occurs in infants less than one year of age.

Cause. This affection is due to the presence in the conjunctival sac of the gonococcus of Neisser, described by him in 1879. (Plate VII.) The contagium is most frequently conveyed by the finger from an active gonorrhœal urethritis or from a gleet; towels, washing utensils, soiled linen, etc., may be the means of carrying it. In all probability the micro-organism is not carried by currents of air. This micro-organism attacks all human conjunctivæ with which it comes in contact, regardless of the condition of the individual.

Description of the Disease, Acute Stage. A period of twelve to thirty-six hours is required after the entrance of the contagium to produce marked disturbances, then the lacrymation is increased, the conjunctiva soon becomes injected, and swelling rapidly advances. Twenty-four hours later the lids have become much swollen, the conjunctiva thickened and deeply injected, and the secretion mucopurulent,

sometimes sanguinolent and mixed with the lacrymal fluid. Burning and gritty sensations are experienced; dull pain in the eye is occasioned by pressure of the lids. In two or three days the height of the acute stage is reached. The swelling of the lids now is often enormous. The eyelids cannot be opened by the patient, and are opened with difficulty by the surgeon. The palpebral conjunctiva is much thickened and velvety, due to a cellular infiltration; the ocular conjunctiva is swollen and often glistening; small ecchymotic spots are sometimes present; chemosis is marked. The secretion, which is yellow, not very thick, and almost entirely free from mucin, flows from beneath the upper lid onto the cheek, matting the cilia. The acute stage continues five to eight days, when it gradually passes into the subacute stage. The tense swelling of the lids subsides and the venous stasis is relieved. The secretion, which is copious, is creamy, the conjunctiva is thickened and thrown into folds and nodules, and the chemosis is less marked. With diminution in the weight and tension of the lids the pain becomes less severe. This stage may last two or three weeks, and recovery then be established; or the disease may pass into a chronic stage, which may continue for weeks or even months.

Severity. The description just given applies to the ordinary cases met with. Cases occur in which the affection is exceedingly light, the discharge scanty and not free from mucus. Some cases are extremely severe, and the swelling of the lids and conjunctiva great. The conjunctiva is pale in hue, from the pressure of the infiltration. Pseudomembranes form on the surface of the palpebral conjunctiva, often closely resembling diphtheria. The severe cases occur most frequently in adults.

Complications. The cornea is involved in about 33 per cent. of the cases occurring in adults; the ultimate impairment of vision varies much; complete destruction of vision may occur. Ulcer of the cornea does not occur ordinarily until the second week of the disease. Gonorrhœal iritis and iridochoroiditis may complicate the attack. Gonorrhœal rheumatism may also result. This complication occurs only in the late stage of the disease. Pseudomembrane forms on the palpebral conjunctiva in perhaps 20 per cent. of the cases that occur in adults.

Diagnosis. Light forms of the disease may be confounded with acute contagious conjunctivitis; severe cases may be mistaken for diphtheria. Microscopical examination of the secretion will serve to make the diagnosis clear in the greater number of cases. In cases that have been under treatment for some time and in the very mild cases it is difficult to find the gonococcus, but patient search is usually rewarded.

Prophylaxis. One who has gonorrhœal urethritis should be cautioned regarding the danger of infecting the conjunctiva. After a gonorrhœal conjunctivitis is established in one eye, care should be observed not to convey the contagion to the other eye. In adults

it is wise to protect the eye either by a carefully applied aseptic bandage sealed at the nasal half with collodion, or, better, a Buller shield may be employed. This consists of a watch-glass which is secured over the eye by means of rubber adhesive plaster. All dressings that come from the eye should be destroyed, and the greatest care should be observed in the disinfection of appliances used. The nurse and others in attendance should be instructed regarding the danger and the precautions necessary. The hands should be washed after touching the eye.

Treatment. In cases that are seen in twenty-four or thirty-six hours after the eye has been infected, it is possible to abort the disease, in a number of cases at least. This is done by thoroughly cleansing the eye, freeing it from all secretion, thoroughly applying a solution of nitrate of silver (1 to 2 per cent.) to the entire surface of the conjunctiva twice in twenty-four hours, and making cold applications to the lids. After three applications the silver may be stopped. The use of boric acid for cleansing the conjunctiva should be continued for a few days, as should also the cold applications. The greater number of cases have progressed too far when seen by the surgeon to permit of abortive treatment. Careful vigorous treatment should be commenced at once. If the lids are much swollen, cold applications should be made constantly. If the lids are not greatly swollen, the applications may be made for three hours at a time, an interval of one hour permitted, and the cold applications resumed. This should be continued until the acute stage has passed, and the frequency and length of time gradually diminished.

METHOD OF MAKING COLD APPLICATIONS. Pledgets of linen one and one-half by two inches square, of three or four thicknesses, or squares of patent lint or absorbent cotton, should be prepared, and, after being moistened, placed on a cake of ice to the number of a dozen or more. (A thin piece of linen may be spread on the ice and the pads laid on the linen.) The pledgets should be changed from ice to eye every one to two minutes, or sufficiently often to keep cool the pledget that rests on the eye. To carry out this treatment requires the constant attendance of two nurses—one for day and one for night duty. It may happen that the eye is kept too cold and the corneal tissue loses its vitality. This calamity may easily be avoided by inspecting the cornea from time to time. In such cases the cornea becomes uniformly hazy, taking on the appearance of ground glass. If the cornea shows the effect of cold, the applications should be made for a few hours at a time, and the intervals lengthened. Heat is not desirable until the gonococci have disappeared; in the later stage of the disease it may be of service.

The eye should be kept as clean as possible by frequent bathing with a solution of boric acid (3 per cent.), or mercuric chloride, 1: 15,000. In cleaning the eye, the lids should be separated very gently and the solution be permitted to enter the eye by dripping from a pledget of cotton, by pouring from an undine, or by a gentle stream from

a pipette. For washing the eye, boric acid, trikresol, potassium permanganate, mercuric chloride or cyanide, formaldehyde 1 : 10,000 to 1: 5000, or hydrogen dioxide (one-third, U. S. P.) may be employed. The peroxide of hydrogen may be employed four or five times daily for this purpose. Potassium permanganate, 1 : 2000, to irrigate the conjunctiva, is efficient.

It has been found advantageous to stuff the conjunctival sac with boric acid ointment (5 per cent. of boric acid vaseline, Wilson) each time after bathing the eye. Applications of a solution of nitrate of silver (0.5 to 2 per cent.) may be made once in twenty-four hours. Solutions stronger than five or ten grains to the ounce are seldom necessary.

Protargol, 20 to 40 per cent., may be applied to the conjunctiva once or twice daily. The continued use of this drug produces a thickening of the conjunctiva that is recovered from but slowly. It may be employed with advantage for a few days during and immediately following the very acute stage.

When ulcer of the cornea is threatened, borated vaseline should be applied at least every two hours to the corneal surface after thorough cleansing of the cornea and conjunctiva. To the vaseline, atropine may be added in the proportion of one grain to the ounce, or atropine in solution, 1 per cent., may be instilled twice daily. If perforation is imminent, paracentesis may be done through the floor of the ulcer. If there is no evidence of congestion or inflammation of the iris, and the ulcer progresses, eserine (0.5 per cent.) may be instilled twice daily. The leucomata and staphylomata and the shrunken globes that follow in some cases should be treated as thought most expedient.

Depletion. If the lids are greatly swollen and the cornea likely to suffer from pressure, a free canthotomy may be performed, which affords depletion as well as release of tension. Critchett's operation, which consists in splitting the upper lid vertically through its entire thickness and stitching the flaps to the brow, restoring the lid by a plastic operation after the disease has subsided, may be resorted to. Scarification of the chemotic tissue may be done in some cases.

Constitutional. The general condition of the patient should be studied, and such measures as are required to maintain the normal vital processes in full vigor should be instituted.

Ophthalmia Neonatorum. Liberally construed, this term may be made to include the purulent or mucopurulent inflammations of the conjunctiva that occur during the first year after birth. Ordinarily the term is applied to those forms of conjunctivitis that appear before the end of the first month after birth.

Cause. All who have made careful bacteriological examinations of the secretion in cases of ophthalmia neonatorum are convinced that the cases that occur before the end of the third day after birth are due almost without exception to the presence of the gonococcus. Cases that occur later may be due to the gonococcus, but not a few

are due to the Koch-Weeks bacillus, the pneumococcus, the Klebs-Loeffler bacillus, or some other form of pathogenic germ or irritating substance.

Method of Infection. We are now concerned with the class of cases that occur in the first few days after birth, and need not mention the modes of infection that produce conjunctivitis later than this period. In almost all of the cases infection undoubtedly occurs during the passage of the child along the genital tract of the mother and just at the time of delivery, due to the entrance of the vaginal secretion into the conjunctival sacs. In rare cases infection takes place ante-partum, the disease being well advanced at birth. In some cases destruction of the cornea has already taken place. Infection by the nurse's hands, unclean washes, and soiled linen may occur after birth.

Description of the Disease. A slight redness of the conjunctiva is usually observed on the second day, and on the third morning the lids are glued together by a small quantity of mucopus. The lids begin to swell, and soon the upper lids become enormously thickened, dusky red, and very tense. They overlap the lower lids, and in the early part of the acute stage seromucopus tinged often with bile pigment oozes from the palpebral fissure. The height of the acute stage is reached on the third or fourth day after the commencement of the disease. Soon the character of the discharge changes to a creamy pus, large quantities of which escape; the conjunctiva becomes greatly thickened, the palpebral portion suffering more than the ocular. The acute stage gradually passes into a subacute condition, in which the swelling of the lids subsides; the conjunctiva although rough, becomes pale and atonic, the discharge a little less creamy and less in quantity. This condition may continue for weeks or months.

Severity. The above is a description of a case of medium severity. Cases of much greater severity are occasionally observed. The onset is more rapid, the secretion serosanguinolent at first; pseudomembrane forms on the palpebral conjunctiva, and the disease resembles diphtheria of the conjunctiva. A number of cases are extremely mild; the onset very slow, and recovery rapid.

Diagnosis. The age of the patient determines the term to be applied to the disease; but it is not always easy to determine the variety of inflammation without a microscopical examination of the secretion. This will serve to relegate each case to its proper category.

Complications. Corneal ulcer, destruction of the cornea, panophthalmitis, iritis, and gonorrheal rheumatism may complicate ophthalmia neonatorum.

Prophylaxis. It has been fully demonstrated that efficient measures taken to prevent the development of ophthalmia neonatorum serve to reduce the percentage from between 9 and 10 per cent. to 0.5 per cent. or less.

Just before and during labor the genitals of the mother should be rendered as aseptic as possible by the use of suitable douches and

washes. Very shortly after the birth of the child the lids should be freed from secretion by wiping with absorbent cotton; the eyes should then be bathed with a weak solution of mercuric chloride, boric acid, or normal saline solution; the lids parted, and one drop of a 2 per cent. solution of nitrate of silver instilled from the end of a glass rod. If more than one drop enters the eye, the solution should be neutralized by washing with normal saline solution. If the reaction is considerable, cold applications should be made to the lids for an hour after the application. This is the method of Crédé, introduced by him at the Lying-in Hospital in Leipzig in 1880. Mercuric chloride, 1 : 2000, may be substituted for the silver.

Treatment. Cold applications are most desirable in the acute stage. They should be made more continuously in severe than in mild cases, but need not in any case be employed so continuously as in adults. In some cases applications should be made for two hours at a time, with intervals of one or two hours. In mild cases, one hour three times daily will suffice. Irrigation with boric acid solution should be done frequently, every half-hour in an ordinary case, to keep the eye free from secretion. Potassium permanganate solution, 1: 2000, or mercuric chloride, 1: 15,000, may be substituted from time to time for the boric acid solution. As soon as the tense, brawny condition of the lid has partly subsided, applications of silver nitrate, 0.5 to 1 per cent., should be made once in twenty-four hours. Protargol, 20 per cent., may be substituted for the silver; but the prolonged use of protargol should be avoided. The applications of the nitrate of silver and the bathing with boric acid solution should be continued until the secretion ceases.

Pathology. The pathology of gonorrhœal ophthalmia and that of ophthalmia neonatorum are very similar. The tissue of the lids is infiltrated by serum, plastic exudation, and small cells. This infiltration is undoubtedly excited by ptomaines produced by the development of the gonococcus in the superficial layer of the conjunctiva. The vessels of the conjunctiva and lids become enlarged and engorged, and a certain degree of venous stasis is produced. The conjunctiva becomes infiltrated with small cells, is much thickened, and the papillæ are enlarged. As the disease subsides all the inflammatory products disappear without leaving a trace, except in the very severe cases, in which there may be loss of conjunctival tissue, and in the very chronic cases, in which nodular masses remain in the conjunctiva and the papillary body remains permanently hypertrophied.

Diphtheritic conjunctivitis is a violent inflammation of the conjunctiva, accompanied by the formation of a pseudomembrane, occasionally accompanying diphtheria of the nose and fauces.

Cause. The Klebs-Loeffler bacillus is the only cause of this form of conjunctivitis, but this micro-organism is soon joined by others, notably the streptococcus and the staphylococcus, which modify the effect of the Klebs-Loeffler bacillus, often increasing the severity of the disease. (Plates VIII. and IX.)

Description. The period of incubation is from twelve to thirty-six hours. Intense swelling of the upper lid, which becomes brawny, dusky red, and very tense, develops rapidly, the firm condition being due to a plastic exudation into the tissue of the lids, venous stasis from pressure imparting the cyanotic appearance. The secretion from the lids is scant at first, being composed of lacrymal fluid, serum, and blood. Very little pus or mucus is seen earlier than the second day after the onset. Gradually the secretion becomes flaky and mucopurulent, containing blood and shreds of fibrin, which character it assumes in the subacute stage, becoming purulent at the end of this stage. The tense swelling of the lids lasts from two to five days, after which the lids become flabby, but remain thickened for one to three weeks. Restoration to the normal condition progresses very slowly.

Pseudomembrane. At the end of twenty-four hours after the congestion of the conjunctiva begins the palpebral conjunctiva is covered with a thin pseudomembrane, which rapidly increases in thickness and extends to the ocular conjunctiva. The pseudomembrane persists until the subacute stage is well established. It rarely attains the thickness of more than one millimetre, and when detached it often presents a perfect cast of the fornix.

Severity. Diphtheritic conjunctivitis may exist without the intense thickening of the lids that has been described, but a pseudomembrane forms and is persistent. The pseudomembrane may be scarcely noticeable and the affection extremely mild. Some cases may be termed "fulminating," so rapid is the onset, so intense the swelling, and so disastrous the result to the cornea.

Pathology. The development of the micro-organism in the conjunctiva appears to cause destruction of the superficial epithelial cells, and so to affect the bloodvessels that a portion of the plasma of the blood escapes into the tissue of the conjunctiva and lids, there coagulating, and producing the tense, firm thickening. The plasma of the blood also escapes onto the conjunctival surface, there coagulating and forming the pseudomembrane. In mild cases where the lid does not become hard coagulation of plastic lymph in the tissues of the lids does not occur. While the formation of pseudomembrane is not pathognomonic of diphtheria, it almost always occurs in diphtheria of the conjunctiva. The greater number of cases known as croupous conjunctivitis are in reality diphtheria; they bear the same relation to the more severe forms that membranous croup does to diphtheria of the nose and pharynx.

Diagnosis. When diphtheria of the conjunctiva is associated with diphtheria of the nose or pharynx, the diagnosis is easily made. However, primary diphtheria of the conjunctiva may occur, and the diagnosis is then not so readily made. The condition may be confounded with gonorrhœa of the conjunctiva, or even with mucopurulent conjunctivitis in rare cases. Bacteriological examination will serve to establish the diagnosis.

Treatment. As soon as it is known that diphtheria of the conjunctiva exists, the patient should be given a hypodermic injection of 1500 to 2000 units of diphtheria antitoxin, making the injections either in the loose tissue in the sides of the abdomen or in the loose tissues of the back. If the pseudomembrane does not begin to soften at the end of twenty-four hours, a second injection of 1500 to 2500 units of the antitoxin may be given.

If the circulation of the lid is not too much interfered with by the swelling, cold applications should be made, as in gonorrhœal conjunctivitis, and as soon as the pseudomembrane is removed nitrate of silver may be applied once daily in the strength of 0.5 to 1 per cent. The eye should be cleansed every hour with a saturated solution of boric acid, a weak solution of potassium permanganate, salicylic acid, or mercuric chloride. Peroxide of hydrogen is of service in the removal of the membrane, if for any reason this is thought advisable. It does no good to remove the pseudomembrane forcibly, unless for the purpose of applying remedies directly to the surface of the conjunctiva (even then it is of doubtful expediency), as the membrane reforms, and the traumatism occasioned opens up new avenues for the entrance of the micro-organisms.

If sloughing of portions of the conjunctiva occur, the endeavor must be made to prevent adhesions between opposing surfaces.

Complications. Ulcer of the cornea, total destruction of the cornea, panophthalmitis, and sloughing of parts of the conjunctiva and lids are the complications met with.

Membranous Conjunctivitis. This is a class of cases in which at the beginning the lids are only slightly swollen and red; there are excessive lacrymation and some mucopurulent secretion; the conjunctiva is slightly thickened. On everting the upper lid, a pseudomembrane is found which extends into the fornix. It is usually not very thick. It may be removed without much force, and on removal discloses a mucous membrane that bleeds only very slightly, but is not deeply injected, nor does it exhibit the characteristics of active inflammation. The pseudomembrane promptly reforms after removal, and may continue to reform indefinitely. Although commonly affecting both eyes, it is sometimes confined to one eye. The individual suffers but little pain; there is but slight photophobia. In many of the cases recovery occurs in from three to five weeks, but in some cases the pseudomembrane persists for as many months in spite of treatment.

Cause. In a number of cases the Klebs-Loeffler bacillus is found. These cases respond readily to treatment. In a few cases the streptococcus is found, and the affection is associated with dacryocystitis. Occurring as an accompaniment of measles, scarlet fever, and influenza (de Schweinitz), the prognosis is not only unfavorable as to vision, but unfavorable to life. Membranous conjunctivitis may accompany impetigo (Morax). The staphylococcus and the pneumococcus have also been found in the secretion in these cases.

Diagnosis. When pseudomembrane occurs in the conjunctiva, it is not always possible to determine the cause. The bacteriological examination will suffice in a number of cases, and the history of the case will determine others.

Treatment. Membranous conjunctivitis due to diseases which have been discussed, viz.: diphtheria, gonorrhœa, mucopurulent conjunctivitis, etc., also accompanying the eruptive fevers, and that due to burns and injury, disappears when the local or constitutional disease is recovered from, or when the effect of the burn or injury has passed away.

In some of the indeterminate forms, which are rare, treatment seems to have little influence. However, cleansing solutions, such as saturated solution of boric acid, salicylic acid in saturated aqueous solution, mercuric chloride (1 : 000 to 1 : 15,000), potassium permanganate (1 : 2000), and hydrogen peroxide, may be employed to keep the conjunctiva free from secretion. Cold applications in the more acute stage may be used intermittently with benefit.

Neighboring disease processes, as dacryocystitis, abscess of the lids, eczema, etc., should be properly treated, and the general system should be put in a healthy condition.

Xerosis epithelialis (xerosis triangularis; xerosis infantilis) is characterized by a lustreless, grayish-white, foamy, greasy deposit on the conjunctiva, which is not moistened by the tears and is very persistent. The disease attacks all individuals except the very old.

Cause. A specific bacillus in this disease was described first by Colmiatti,[1] and carefully studied by Leber[2] three years later, and termed by him the diplobacillus of xerosis. The bacillus is short, and often appears in pairs joined end to end. One of the members is often broader at one end than the other (clubbed); the cheesy secretion contains multitudes of the bacilli almost in pure culture.

Description and Symptoms. A receptive condition of the system appears to be necessary to permit the development of the disease. When infants are attacked, it is always the marasmic infant; the robust never contract the disease. Children and adults always give a history of malnutrition, most often because of scanty food, with scarcity of fresh vegetables and fresh meats—those who are confined in barracks, prisons, or who work remote from a base of plentiful food supplies, as in mines, or railroads, or on plantations. In infants the lids become slightly swollen, and a thin flaky secretion escapes; the infant is but little disturbed by the condition present. On everting the lids, the conjunctiva in the fornices is more or less covered by the characteristic secretion. The secretion may extend over the whole conjunctiva and cornea. Both eyes are affected. In children more than one year of age the secretion may show itself first either on the palpebral or ocular conjunctiva; in adults almost always on the ocular conjunctiva, the patch occupy-

[1] Cong. périodique inter. de Ophth. Annexes, May 28, 1880.
[2] Graefe's Arch., 1883, Band xxix., iv., S. 225.

ing the conjunctiva in the horizontal meridian on both sides of the cornea, usually triangular in shape, the base being next to the margin of the cornea. The sensation to the adult is that of a dry substance on the conjunctiva. One patient spoke of it as his "dry patch." Slight irritation of the conjunctiva is noticeable about the margin of the patch. In children and in adults a condition of hemeralopia obtains. The disease is not a local one. In a number of autopsies that have been made, the bacillus was found in the parenchyma of the liver, spleen, kidneys, and pancreas.

Duration. In infants this disease lasts until death. In adults the secretion persists for months, and in many cases for years.

Complications. In infants the cornea is deprived of nutrition and sloughs. All infants under one year of age die. In children and adults the cornea may become involved, the patches of exudation gradually advancing from the margin of the cornea, or appearing in small islets slowly encroaching on the pupillary area. Years may pass before the pupillary area is completely covered. Fortunately, in the greater number of adults and children the cornea does not become involved, provided suitable treatment is instituted.

Diagnosis. The condition cannot be mistaken for anything else after the clinical picture is recognized by the surgeon.

Pathology. Aside from the presence of the bacilli, the superficial epithelial layers undergo fatty degeneration and death, and are cast off. The oil globules in the cells are very minute. The secretion consists of these degenerated epithelial cells, some leucocytes, and the bacilli. On examining the conjunctival tissue little change is found, except a slight increase in size of the bloodvessels, some small-cell infiltration, and the thickened and superficially degenerated epithelial layer.

Treatment. In infants it is of no avail. In adults the local treatment consists in the use of antiseptic lotions and washes, and of ointments, such as bichloride vaseline (1 : 5000), the use of powders —calomel, iodoform, aristol—the powders to be dusted on the affected area after the secretion has been gently wiped off. Unless the local treatment is supplemented by a nutritious and varied diet, a cure cannot be expected. Suitable tonic remedies should supplement the diet.

Phlyctenular conjunctivitis (conjunctivitis eczematosa) is characterized by the appearance on the bulbar conjunctiva of one or more small nodular elevations, which are situated at the apex of a triangular leash of vessels, the base of the leash being directed toward the fornix conjunctivæ.

Cause. The writer has felt justified in including this affection among those that are caused by a specific micro-organism, because of the researches of others as well as of himself. If an unbroken phlyctenule be carefully rendered aseptic externally and the contents of the phlyctenule conveyed to a tube of nutrient agar, a culture of the staphylococcus will invariably be obtained. The same

is true of the nodule of eczema. Similar nodules may be produced by introducing the staphylococci beneath the epithelium in suitable subjects. Pustular blepharitis marginalis and moist eczema are frequently accompanied by phlyctenular conjunctivitis or keratitis; they are undoubtedly sources of infection. Phlyctenular conjunctivitis is most frequent in children of the poorer classes who have inherited taints or are tubercular, or who suffer from malnutrition accompanied by lymphadenitis, by moist eczema on some part of the body, particularly on the head, face, and ears; eczematous rhinitis, etc. (Fig. 135.)

Fig. 135.

Phlyctenular conjunctivitis in a scrofulous subject. (Dalrymple.)

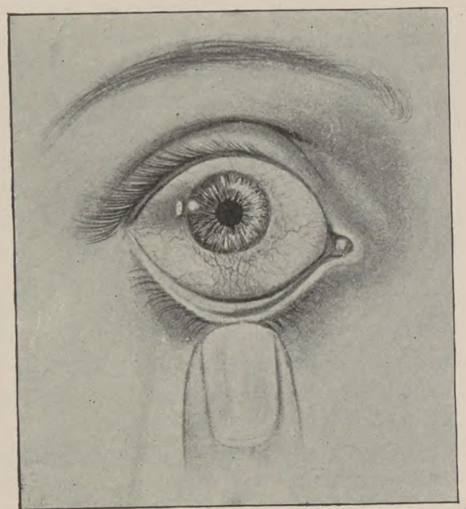
Adults are not exempt, but they are rarely attacked. Occasionally an apparently robust individual is affected, but in these cases blepharitis marginalis or a patch of moist eczema is present or has preceded the attack of phlyctenula. Acute conjunctivitis, the exanthemata, and debilitating illness of any kind predispose to eczematous conjunctivitis.

Description and Course. In the early stages small translucent nodules appear at the limbus conjunctivæ or on the bulbar conjunctiva. (Fig. 136.) The bloodvessels of the conjunctiva radiating from the nodule become injected. The nodules may be single or multiple. Soon the apex of the nodule softens and disappears, and the contents of the vesicle take on a yellowish appearance. The softening

progresses until the nodule has reached the level of the conjunctiva, when the ulcer becomes clean; epithelium is developed on its surface, and recovery occurs without leaving a scar. Recurrences are the rule. The process from the first stage, the stage of efflorescence (Fuchs), to complete recovery requires eight to fourteen days.

When but one or two nodules are present, the redness of the conjunctiva is but partial, and is confined to the vicinity of the nodule. There is but slight increase in lacrymation and little irritation; no photophobia. When many nodules exist, the redness may extend to the palpebral conjunctiva, the lacrymation and secretion may be much increased, and some photophobia may be experienced.

FIG. 136.



Phlyctenular conjunctivitis. (DALRYMPLE.)

Diagnosis. Phlyctenular conjunctivitis may be confounded with herpes of the conjunctiva, pinguecula, lymphangiectasis, and vernal catarrh, but the history of the case will serve to make the differential diagnosis.

Pathology. The elevation or nodule is composed of an accumulation of small cells resting on the basement membrane and causing an elevation of the epithelium. The bloodvessels at the base of the nodules are engorged and enlarged, and there is a scant small-cell infiltration in the surrounding tissues.

Treatment should be local and constitutional.

LOCAL. A cleansing aseptic wash should be used to bathe the eye three or four times daily. An ointment of the yellow oxide of mercury (1 per cent.) should be put into the eye twice daily. After the nodule has been converted into an ulcer, calomel may be dusted over the affected area once daily, if the patient is not taking iodine.

INTERNAL. Suitable tonic remedies should be given. Small and frequently repeated doses of calomel (gr. $\frac{1}{20}$ to $\frac{1}{10}$, three times daily) continued for some weeks, if no disturbance of the bowels is occasioned, are of much value. The nasal and pharyngeal cavities should be properly treated.

Tuberculosis of the conjunctiva may be primary or secondary; primary when it originates in the conjunctiva itself, which is not the rule, and secondary when it proceeds from a tubercular focus in some other part of the body.

In *primary tuberculosis* of the conjunctiva, which is the more acute form, the tubercle bacilli enter the conjunctival tissue through wounds of the conjunctiva, sometimes due to operative procedure. According to Valude, tubercle bacilli cannot penetrate the intact epithelial layer.

Course and Symptoms. Within a week or ten days after the entrance of the bacilli the conjunctiva in the vicinity of the place of entrance becomes injected, and numerous small nodules, miliary tubercles resembling trachoma granules, appear. This may occur on the ocular or palpebral conjunctiva. The lids become slightly swollen; a not very copious mucopurulent secretion forms; there are irritation and photophobia. The nodules rapidly increase in number, and may coalesce in places. Within a few days after the onset the preauricular and inferior maxillary glands on the affected side become swollen and may go on to suppuration. The tubercular process may extend to other parts of the body. The disease runs a very protracted course, and may involve the cornea and result in loss of vision. Some rise of temperature accompanies the early stages of this form of tubercular infection.

Diagnosis. This form of tuberculosis may be mistaken for acute trachoma and for Parinaud's conjunctivitis. Its monocular character and the marked involvement of the lymphatics on the affected side will be sufficient to exclude trachoma. Examination of a section of the nodule will disclose the bacilli, differentiating it from Parinaud's disease.

Treatment. Aside from early excision of the affected tissue little can be done. Appropriate constitutional treatment and attention to the symptoms as they arise are all that is possible.

The *secondary form of tuberculosis* is chronic from the onset. It is the disease formerly known as lupus vulgaris, and is most frequently due to extension from the nasal mucous membrane by way of the lacrymal passages. It is characterized by the appearance of irregular shallow ulcers on the palpebral or ocular conjunctiva (more frequently on the palpebral conjunctiva), with raised edges and grayish, uneven floors, often presenting granulation tissue. The surrounding conjunctiva is but slightly injected, the lids are slightly thickened, and a small amount of rather thin, flaky mucopus is present; there is little pain, and the disturbance to the patient, except from the presence of the mucus, is slight. The preauricular glands on the affected side

are enlarged, but there is little tendency to suppuration. An affected area may eventually be occupied in whole or in part by cicatricial tissue. In old cases the conjunctival sacs may be entirely obliterated by the cicatricial process, the cornea may become involved, and vision be lost. Years may pass with but little change, but the tendency is to slow and steady progress.

Diagnosis. Tuberculosis of this form may be mistaken for epithelioma, or chalazia which open on the conjunctival surface. Study of a section of the tissue with the microscope will suffice to make a diagnosis.

Pathology. The change in the tissue in the first form is such as is found in miliary tuberculosis. In the second form the margins of the ulcers are made up of a small-cell infiltration of the conjunctival tissue, with increase in vascularity. In the tissue of the wall of the ulcer the tubercle bacillus is found.

Treatment. Excision of the diseased parts, thorough scraping of the base, and frequent inspersion of iodoform will produce good results. The ulcerated areas may also be destroyed by means of the cautery.

Leprosy of the conjunctiva may occur as a primary infection, but it is secondary to leprosy in other parts of the body in by far the greater number of cases. Morrow[1] cites a case in which a leprous tubercle appeared on the eye and was mistaken for sarcoma. Cutaneous tubercles followed. A sclerosed, anæsthetic condition of the conjunctiva follows the appearance of leprous nodules on the conjunctiva. This process may invade the cornea. Irregular pterygia are sometimes produced. An infiltration of the cornea unattended by pain is observed in the later stages of this affection. A mild persistent irritation of the conjunctiva, with slight redness and increased lacrymation, has been observed by the writer in cases of leprosy. Fuchs mentions iritis and cyclitis as accompanying leprosy of the ocular conjunctiva and cornea.

Syphilis of the conjunctiva manifests itself in a number of forms corresponding with the stage of the disease. Chancre, papillary syphilide, copper-colored spots, mucous patches, gummata, nodular syphilides, and syphilitic ulcer may appear in the conjunctiva.

Chancre appears most frequently on the tarsal conjunctiva, extending to the margin of the lid, but may occur on the retrotarsal folds or ocular conjunctiva. It possesses an indurated base much resembling a piece of parchment. Considerable irritation is produced by the chancre and a rather profuse mucopurulent secretion accompanies it. The elevated mass sometimes disappears without ulceration; but usually the apex of the chancre softens and disappears, and a shallow ulcer with indurated sides and base is present. The papillary syphilide is not common. It accompanies papillary syphilides on the face and lids. Copper-colored spots are not of common occurrence,

[1] System of Diseases of the Skin, Dermatology, vol. iii. p. 587.

but have been observed to accompany the same conditions on the skin. Mucous patches occur in the second and early tertiary stage. They are seen most commonly at the margin of the lid, extending onto the tarsal conjunctiva, but may occur at any part of the membrane. They are slightly elevated, with an even, grayish, furfuraceous surface. Gummata affect the conjunctiva of the lids, or the bulbar conjunctiva near the limbus. They appear as reddish nodules, having a purple hue in some cases, and in some a light-colored apex. The base of the gumma is injected. The gumma develops rapidly and involves the underlying structure. If not properly treated, it eventually breaks down, producing a deep ulcer, the healing of which is long delayed. It leaves a deep cicatrix. Multiple gummata are seldom seen. Gumma may be mistaken for sarcoma. Nodular syphilides are less destructive and pursue a much less violent course than the gummata. They appear as deep-red nodular masses with little tendency to break down. They may eventually disappear without leaving a trace. They may be multiple. Syphilitic ulcer is probably in all cases the result of the breaking down of a gumma or a tubercular syphilide.

Prognosis. If recognized early, the prognosis, with suitable treatment, is favorable in all cases.

Treatment. Systemic treatment must be active, sufficient, and long continued. Locally the eye should be cleansed frequently with a solution of mercuric chloride (1 : 10,000). An ointment of mercuric chloride (1 : 5000 in vaseline) may be placed in the eye after each cleansing, or at least three times a day.

Amyloid disease of the conjunctiva is a very rare affection. It is characterized by the appearance of waxy, translucent, polypoid masses which commonly spring from the lower fornix, but may involve the entire conjunctiva, converting it into large folds which overlap the cornea and greatly obstruct vision. The tissue is almost devoid of bloodvessels and is very friable. Adults only are attacked. The disease is apparently a purely local one.

Pathology. The masses are found to be made up largely of lymphoid cells, which in parts near the surface undergo a change, converting them into a homogeneous mass, which, in the greater number of cases, give the starch reaction to the iodine test.

Treatment. Excision of the masses is necessary. Recurrences are the rule. If the bases are treated by superficial cauterization, return is less liable to take place.

Chronic Conjunctivitis (Chronic Ophthalmia). A thickened, injected condition of the conjunctiva sometimes follows an acute conjunctivitis; accompanies blepharitis marginalis in old people particularly; depends on partial or complete closure of the canaliculæ or eversion of the puncta, and trophic or hypertrophic rhinitis. Errors of refraction and muscle anomalies serve to perpetuate the condition. In old people a flabby, slightly congested, swollen condition of the conjunctiva exists, associated with enlargement of the

caruncle. These cases are almost always accompanied by slight mucopurulent discharge.

Treatment consists in correcting all conditions that stand in a causative relation to the conjunctivitis. The nasal and lacrymal passages should receive careful attention. The conjunctiva itself should be brushed with a solution of nitrate of silver (1 to 2 per cent.) if secretion is present, and it should be kept free from secretion by bathing with a simple cleansing solution.

Egyptian Ophthalmia. This term has been used indiscriminately to describe all forms of ophthalmia that affect large numbers of individuals, especially the forms that appear epidemically. The term has been made to include acute contagious conjunctivitis, gonorrhœal conjunctivitis, and trachoma. The last-named disease has been most generally indicated when the term was employed.

Atrophy of the Conjunctiva (Xerophthalmia). This condition, not accompanied by the presence of the xerous bacillus, occurs in a number of forms:

(*a*) Cicatricial, as from trachoma; extensive burns of the conjunctiva, as from lime or from liquid ammonia. In cicatricial xerosis the ducts of the lacrymal glands are obliterated and the gland itself atrophies. In addition, the character of the conjunctiva is entirely changed, so that no mucus or other lubricating fluid is secreted from it. The cornea becomes opaque, and vision is reduced to perception of light.

(*b*) Xerosis from constant exposure to the air, as in ectropium and in lagophthalmos. In cases of this kind the exposed conjunctiva and cornea take on a cutaneous appearance; the epithelium becomes thickened, corneous, and dry, a provision on the part of nature to protect the deeper layers from desiccation. In this form the remedy lies in the operative procedure necessary for the restoration of a proper protection to the exposed parts.

Toxic Conjunctivitis. This term is applied to the forms of conjunctival irritations that are caused by the chemical action of certain substances. Of these, may be mentioned the mydriatics, the myotics, chrysarobin, calomel, the dust from aniline dies, bites of insects, caterpillar hairs, fumes from formalin, menthol etc., intense light, as from the electric arc light, the reflection of sunlight from the snow.

Atropine produces two forms of disturbance:

(*a*) After long use of a non-sterile solution the conjunctiva becomes hyperæmic and follicles develop in the fornix and tarsal conjunctivæ. There is a scanty mucopurulent discharge. The picture is one of mild trachoma in the early stage. The cause of this form of conjunctivitis is probably bacterial infection, the bacteria being carried into the conjunctival sac with the solution.

(*b*) Six to twelve hours after the instillation of a few drops of a solution of atropine into the eye the lids become swollen and brawny and the conjunctiva injected. There are excessive lacrymation, a sensation of heat, and much irritation. Hyoscyamine, duboisine, and

homatropine sometimes produce this disturbance also, but in less degree. The effect is believed to be due to idiosyncrasy.

The **treatment** of the first form consists in discontinuing the atropine, or using sterile solutions, cleansing the eye frequently with a saturated solution of boric acid, and using suitable astringents. Of the second form, in discontinuing the use of the atropine.

Eserine solutions sometimes cause irritation of the conjunctiva.

Chrysarobin, used in the form of an ointment on the skin in psoriasis, may produce intense irritation of the conjunctiva. Calomel, if dusted on the conjunctiva when the patient is taking an iodide, results in local ulceration and marked irritation of the conjunctiva.

Cocaine when applied to the conjunctiva produces at first a contraction of the bloodvessels, but as its effect wears away there is an engorgement of the vessels which stimulates and irritates the conjunctiva, producing a mild form of conjunctivitis.

The *bites of insects* commonly occur on the lids, and the affection of the conjunctiva, which sometimes becomes greatly œdematous, is due to extension of the irritation.

Treatment. All of these forms of conjunctival irritation subside in a few days if the cause is removed and simple cleanliness observed.

Ophthalmia Nodosa This condition is due to the presence of caterpillar hairs on the conjunctiva. The affection may extend to the cornea, and even to the iris. The nodules are yellowish, semitranslucent and have been compared to tubercles. On excising the nodules and examining them under the microscope, Pagenstacher found the hairs of caterpillars.

Abscess of the conjunctiva is of rare occurrence. It forms in the subconjunctival tissue and is almost always traumatic.

Treatment should be that as of abscesses in other parts of the body.

Ecchymosis of the conjunctiva is due to the escape of blood beneath the conjunctiva from whatever cause. It produces bright- or dark-red patches, and may affect all but the tarsal conjunctiva.

Treatment. The blood becomes absorbed slowly. Bathing with hot solutions hastens absorption.

Chemosis. This is a condition characterized by swelling and thickening of the ocular conjunctiva; the swelling at the corneal margin forming a raised wall, producing a shallow circular pit, of which the cornea forms the floor.

Pathology. Chemosis is more than simple œdema in many cases. Particularly is this so when the chemosis is the result of a slow inflammation of the cornea, iris, ciliary body, and choroid. Sections of chemotic tissue in acute cases show little but a distention of the conjunctival tissue by serous or seroplastic infiltration; but in the subacute or chronic forms there are a dense infiltration of small cells and an increase in connective-tissue elements and in the size and number of the bloodvessels.

Emphysema of the conjunctiva is characterized by puffiness of the conjunctiva, with little injection, and usually with the appearance

of small circular pale points just beneath the conjunctiva, which indicate the presence of air in the tissue. On pressing the conjunctiva, there is a faint crepitation, and the circular points change their position. The condition is due to the entrance of air into the subconjunctival tissue as a result of traumatism, the injury that most frequently produces it being fracture of the lacrymal bone. On blowing the nose violently, air sometimes finds its way into the orbital and subconjunctival tissues, causing them to puff up.

Treatment. The air in the tissues disappears by absorption in a few days.

Injuries to the Conjunctiva. It frequently happens that foreign bodies enter the conjunctival sacs. These impinge first upon the globe, and are then brushed downward by the upper lid. They may remain in the lower conjunctival pouch, but often are caught on the tarsal conjunctiva of the upper lid, from which they may be removed on everting the lid. When lodged on the conjunctiva of the upper lid, they are found most commonly in the shallow groove which lies immediately above the inner angle of the margin of the lid (sulcus marginalis or sulcus tarsalis). Rarely the foreign body lodges in the retrotarsal fold. Slight pressure backward on the globe after the lid is everted will serve to expose this fold, when the foreign body may be removed. Bits of steel are sometimes imbedded in the conjunctiva; they may be removed by the ordinary surgical procedures. Grains of powder that are deeply imbedded need not be disturbed, as they produce no irritation after the wound made by their entrance has healed.

Wounds. These may be associated with extensive injuries to orbit and lids, or may be simple lacerations. Under favorable circumstances they may be cleansed and the margins of the wounds approximated by sutures.

Burns are occasioned by the entrance of flame, glowing wood or metal, powder, ashes, steam, hot water, molten metal, acids, alkalies, nitrate of silver, and other substances.

Treatment. When the burn is occasioned by thermal agencies alone, the treatment should be by means of bland oils or vaseline, to be placed in the conjunctival sac every two or three hours. If molten metal has entered the eye, all of the particles should be removed as early as possible, and treatment instituted as above outlined.

Burns from acids, if recent, should be treated by means of a weak alkaline solution (bicarbonate of sodium, sodium hydroxide, or very dilute ammonia); subsequently the conjunctiva may be well washed with water, and the oils then employed.

Burns from lime and ammonia should be treated by first removing all particles of lime or mortar by means of forceps or cotton pledgets, then by washing thoroughly with oil, and subsequently dropping in simple syrup made from cane sugar, as this forms an insoluble combination with lime. Oil or vaseline, medicated with boric acid (5 per cent.), may then be used until healing takes place.

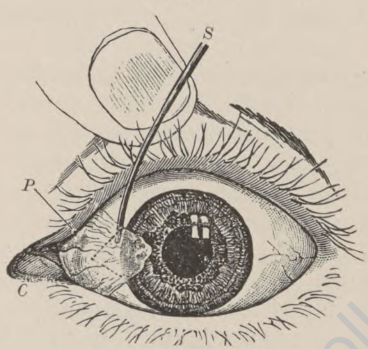
Burns from lime and ammonia produce a pale condition of the conjunctiva, and usually a white eschar where the caustic has come in direct contact with the tissue, or where the effect has been concentrated the cornea may be slightly hazy. The prognosis is usually worse than is at first supposed.

The result to be feared in burns of the conjunctiva is symblepharon. To prevent this, the surfaces should be separated daily by means of a probe, and oil interposed. If the burn is extensive, a pledget of gauze or pad of cotton dipped in oil may be kept between the opposing burned surfaces.

Argyria (Argyrosis) Conjunctivæ. Long-continued use of nitrate of silver on the conjunctiva produces a discoloration of the mucous membrane, due to the deposition of the carbonate or albuminate of silver in the tissue of the conjunctiva (elastic fibres, Fuchs). The stain varies in color from a light ochre to a very dark brown. It is indelible. A solution of hyposulphite of sodium or of potassium iodide in the strength of 1:10 in water has been suggested for its removal.

Pinguecula. In many individuals who have been exposed to dust or high winds there appears in the ocular conjunctiva, on the nasal side of the cornea, later on the temporal side, both in the horizontal meridian, a yellowish elevation measuring two or three millimetres in diameter. This tumor is at first movable on the sclera. It consists in a thickening of the conjunctiva, particularly in an increase of the elastic fibres, and the deposition of numerous minute hyaline particles. The yellow elastic tissue and the hyaline bodies give it the yellow color. If the tumor causes annoyance by its appearance or by becoming inflamed, it may be removed by excision.

FIG. 137.



Pterygium. A sound, *S*, is carried beneath the edge of the pterygium, *P*. The dotted line shows the way in which the section is made in removing the pterygium. *C*. Caruncle. The adjacent plica semilunaris has been flattened out by the tensive force of the pterygium, and is hence invisible. *P*. Upper punctum lacrymale. (FUCHS.)

Pterygium. Pterygia may be classed as regular and irregular. The regular form of pterygium consists in a triangular fold of mucous membrane occurring on the ocular conjunctiva in the horizontal meridian, its base being at the canthus, its apex at the margin of or on the cornea. (Fig. 137.) The blood-vessels enter at the base, diminish in size, and converge at the apex. Pterygium occurs in individuals of middle and advanced life, most frequently developing on the nasal side of the cornea; it may also appear on the temporal side. Pterygia may be either progressive or

stationary. In progressive pterygium the fold of mucous membrane has a fleshy appearance and its vessels are pronounced. The apex of the growth is preceded by a grayish zone, one to one and one-half millimetres wide, which is very slightly raised at the margin of the apex of the growth. In non-progressive pterygium the growth is pale and flat, and the grayish zone is narrow, and is flat or slightly shrunken, appearing like a cicatrix.

Pterygium may advance until it passes the pupil; it may stop at any point on the cornea short of this.

Irregular or pseudopterygium is the result of burns or ulcers of the cornea. They have the same general shape as the regular pterygia, but the apex is often very irregular in contour, taking on the shape of the margin of the ulcer farthest removed from the limbus of the conjunctiva. The conjunctiva at the margin of the cornea corresponding to the ulcer becomes swollen, and a fold of chemotic conjunctiva becomes agglutinated to the floor of the ulcer. As the ulcer closes and cicatricial tissue forms, the mucous membrane is drawn onto the cornea, at the same time becoming hypertrophied. The pterygium never progresses beyond the cicatricial floor of the ulcer.

Regular pterygia are undoubtedly due to irritation of the conjunctiva in its most exposed part, corresponding with the palpebral fissure, by particles of dust and various minute bodies that impinge on the ocular conjunctiva. This irritation results first in the formation of pinguecula; extending, it produces the pterygium.

Pterygium is prone to become inflamed because of the lodgement of noxious germs or irritating particles in the folds of the mucous membrane. These inflammations may lead to ulcer of the conjunctiva, ulcer of the cornea, and serious damage to vision.

Diagnosis. Pterygium cannot well be mistaken for anything else.

Treatment. The cautery may be used to destroy the head of the growth, or to cut off its nutrition by making one or two deep grooves across the neck of the pterygium.

The operative procedures resorted to most frequently are excision, transplantation, and strangulation.

Excision. The neck of the pterygium is grasped by the fixation forceps and pierced close to the limbus by a sharp cataract knife, the knife passing just below the tissue of the pterygium and parallel with its surface, the edge of the knife being directed toward the cornea. By a to-and-fro motion the knife is made to pass beneath the head of the pterygium, dissecting it from the cornea. Instead of this procedure, a thin strabismus hook may be passed through the incision made beneath the neck of the pterygium (Prince's method), and the head of the pterygium torn from the cornea. The body of the pterygium is now dissected from the underlying tissues for a distance of three to six millimetres, and a diamond-shaped piece excised, the inner apex lying at the caruncle. The conjunctiva is now loosened from the underlying tissues above and below, and the edges brought together by sutures, covering the defect as far as the

margin of the cornea. The corneal defect becomes covered with epithelium in a few days and healing progresses satisfactorily, leaving some opacity.

Transplantation (Knapp). Instead of excising the pterygium after having dissected the growth toward the caruncle, the corneal tissue may be removed from its head and a suture passed through the apex of the pterygium. A straight incision may now be made in the lower bulbar conjunctiva, extending from the margin of the wound about four millimetres from the cornea, downward, and slightly outward, toward the fornix, sufficiently long to accommodate the free part of the pterygium. The suture through the apex of the pterygium is now passed through the conjunctiva at the apex of the last incision, and the head of the pterygium drawn into the space and there fixed. This directs the tissue of the pterygium into the lower fornix. All defects other than the corneal are now covered by suturing the edges of the conjunctiva. Instead of transplanting all of the pterygium below, the body of the pterygium may be split—one-half may be transplanted below, one-half above.

Strangulation. The neck of the pterygium is grasped by the fixation forceps and slightly raised. A suture with a needle near both ends is employed, one needle passing upward beneath the neck of the pterygium at the corneal margin, the other needle passing upward beneath the neck of the pterygium three millimetres from the corneal margin. The ends of the suture are not drawn through, but the loop holding each needle is cut, liberating the needles and forming three sutures. The suture at the corneal margin is tied tightly over the neck of the pterygium, as is also the suture nearest to the caruncle. The middle suture encircles the base of that portion of the pterygium lying between the two end sutures. The middle suture is now tied, producing strangulation of a section of the pterygium. The sutures are permitted to remain until they come away spontaneously. Deprived of nutrition, the head of the pterygium atrophies and disappears, leaving only an opacity.

Recurrences. These are not infrequent after removal by excision, but are very infrequent after transplantation and strangulation.

Lymphangiectasis consists in dilatation of some of the lymph channels of the ocular conjunctiva. It appears as slightly elevated, transparent vesicles, usually associated in chains, very superficially situated in the outer or inner half of the bulbar conjunctiva. The vesicles are irregular in shape and vary in size, seldom exceeding a diameter of three millimetres. The vesicles may be readily moved over the underlying tissue. They produce no irritation and are not a source of pain. The vesicles are due to interference with the lymph stream by obstruction. Elderly individuals are more frequently affected.

Treatment. The vesicles may be excised, or they may be very satisfactorily destroyed by means of the fine galvanocautery point. Removal is necessary only for cosmetic purposes.

Vascular growths in the conjunctiva are seldom primary, but are commonly extensions from the tissues of the lids. They appear as arterial growths, when they are of a bright-red color, slightly elevated; as venous growths (cavernous angioma), located deep in the conjunctiva, dark purple in color; or as telangiectatic growths—bright-red patches in the conjunctiva. All these conditions may be present in the same growth. Vascular growths are congenital. They tend to increase in size. Early removal is advisable. (See Removal of Vascular Tissues of the Lids.)

Polypi. The occurrence of polypi on the conjunctiva is probably always associated with ulcerative processes of a more or less chronic nature in the conjunctiva. Wounds of the conjunctiva that do not properly close, syphilitic or tubercular ulcers, sinuses from chalazia opening onto the conjunctival surface, sinuses from areas of orbital necrosis, all give rise to the development of polypi. The irritation from wearing an artificial eye may result in the development of polypi.

Pathology. Polypi of the conjunctiva are composed of myxomatous tissue with more or less small-cell infiltration, according to the degree of irritation.

Treatment. Removal and correction of the conditions favoring their formation.

Benign Tumors. Those that develop primarily in the conjunctiva are adenoma, fibroma, granuloma, lipoma, myxoma, osteoma, papilloma, simple cystic tumors, and those due to cysticerci and echinococci.

Adenoma occurs rarely as an extension from the tarsus or as a development from lacrymal glandular tissue, or from the caruncle.

Fibromata are usually the result of chronic conjunctivitis, particularly of vernal catarrh.

Granuloma develops from the base of an ulcer and from wounds.

Lipoma occurs in the fornix in the shape of a soft yellowish mass.

Myxoma. The most common form is polypus.

Osteoma is of extremely rare occurrence. (See Congenital Conditions.)

Papilloma. Tumors of this nature exist as small multiple papillæ, forming soft, pale-pink, villous masses. They may develop from any part of the ocular or palpebral conjunctiva, but are seen most commonly on or near the caruncle. Papilloma is not infrequently mistaken for granulation tissue. To avoid recurrence, removal should be thorough.

Simple cystic tumors appear in the palpebral conjunctiva after chronic conjunctivitis, after plastic operations on the conjunctiva, and after operations on the ocular muscles. They usually form as a result of the invagination of epithelium. The treatment is excision.

Cysts due to entozoa are very rare. Cysticercus cysts are large. If the walls are thin, the head is visible as a white spot at some part of the cyst. They are easily removed by splitting the conjunctiva

over the cyst and turning the cyst with its thin capsule of connective tissue out of the wound.

Echinococcus cysts are large. They develop slowly, and may extend into the orbit and produce marked exophthalmos. Daughter cysts and hooklets may be found as part of the contents of the cyst.

Malignant Tumors. Epithelioma and sarcoma are the most common. Russell describes a rare growth known as cylindroma. It is probably a form of sarcoma.

Epithelioma affecting the conjunctiva is much more frequently secondary; that is, an extension of a growth originating in the lids. When it is primary it springs from the limbus and extends onto the cornea. It is of slow growth, appearing as a slightly raised patch with a roughened grayish surface.

Sarcoma, primary in the conjunctiva, is almost always pigmented. It occurs where pigment is often normally present, as at the limbus, where it is more frequently met with, and in the conjunctiva of the lids. It has been observed at the caruncle. Sarcoma of the conjunctiva may remain quiescent for years, suddenly taking on activity and terminating fatally in a short time. Metastasis to the preauricular and cervical glands and to remote parts of the body may occur.

Treatment. Complete excision is the only treatment that is of value.

Lupus erythematosus, when it affects the conjunctiva, appears as small irregular plaques covered with grayish masses of exudation and superficial cicatrices, sometimes with punctate excoriations. Lupus erythematosus of the face accompanies the conjunctival affection. The disease progresses slowly, and is accompanied by slight irritation and increased lacrymation.

Etiology is not well understood. When disease of the face accompanies that of the conjunctiva the diagnosis is readily made.

Treatment is of no avail.

Acne of the Conjunctiva. This condition sometimes accompanies acne nodosum of the face. The ocular conjunctiva usually is affected. The condition resembles phlyctenular conjunctivitis closely.

Affections of the Caruncle and Semilunar Fold. Inflammation of the caruncle may be due to infection of one of the glands of the caruncle, resulting in the formation of an abscess. The abscess may be incised, or it may be permitted to open spontaneously, when it will readily heal. The hairs of the caruncle may become a source of irritation. Epilation or extirpation is the remedy.

Papilloma may develop on the caruncle, where it presents the same characteristics as when it appears on other parts of the conjunctiva. Congenital telangiectatic growths may appear in the caruncle.

Encanthus is a term applied to any enlargement of the caruncle. In all forms of conjunctivitis there is enlargement of the caruncle, which disappears as the inflammation of the conjunctiva subsides.

Cystic enlargement is sometimes observed. *Chalky deposits* may occur in the glands of the caruncle, causing enlargement. *Adenoma* may develop. When the enlargement is due to development of sarcoma or epithelioma, the term *eucanthus maligna* is applied.

Treatment in all cases of enlargement from the development of new-growths should be excision.

Symblepharon. Cicatricial union of the palpebral to the bulbar conjunctiva is termed symblepharon. It occurs after burns, injuries, and some operative procedures, and as the result of purulent conjunctivitis, pemphigus, and trachoma. The bands of cicatricial tissue may extend to the cornea. Should the union between the lids and globe be complete, the condition is termed *symblepharon totale*. If the union extends from the bottom of the fornix, partially uniting the lid to the globe, it is termed *symblepharon posterius*. When the union of the lid to the eyeball is such that the cicatricial band does not extend to the bottom of the fornix, the condition is termed *symblepharon anterius*

The **treatment** of symblepharon is surgical. In symblepharon anterius, carefully dissect the lid from the eyeball, and, if the adhesion is not extensive, the surfaces may be separated daily until cicatrization has taken place; a pledget of cotton soaked with olive oil may be interposed between the raw surfaces and permitted to remain until healing occurs. The bridge of tissue may be ligated and the ligature allowed to slough through.

In extensive anterior symblepharon, the defect in the bulbar conjunctiva may be covered by dissecting the conjunctiva at the border of the defect from the underlying tissue, making sliding flaps from both sides and uniting the margins of the conjunctiva over the defect in the ocular conjunctiva. With an epithelial surface opposed to the defect in the conjunctiva of the lid the defect in the lid will cicatrize without adherence to the globe.

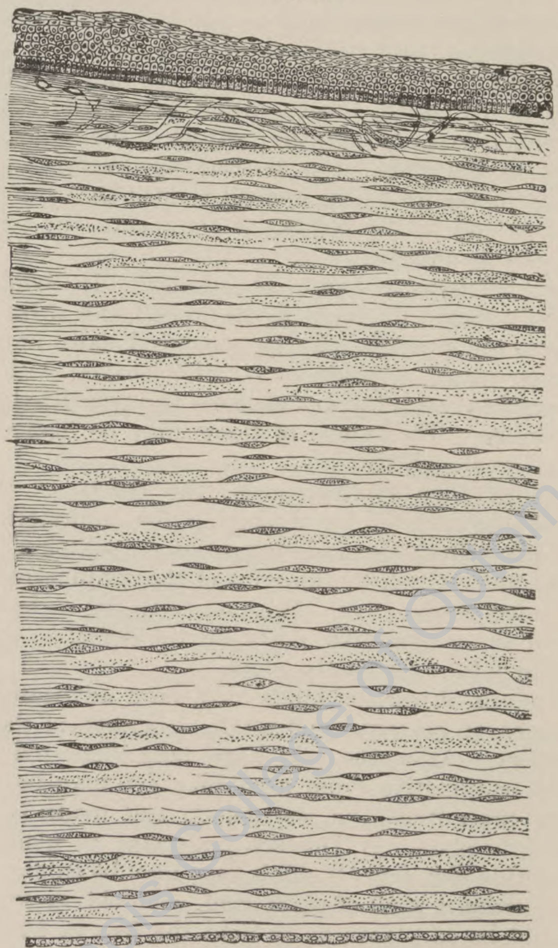
Plastic operations of various kinds have been advocated for correcting symblepharon posterius and totalis; but none of them is perfectly satisfactory. After the lids have been dissected from the globe a flap of conjunctiva from a rabbit's eye may be conveyed, with antiseptic precautions, to the defect, and made to cover it, being stitched into place. A Thiersch graft may be made to cover the defect, or a thin skin flap (Wolffe's flap) may be employed. After the flap is in position a shell of glass, lead, celluloid, or some similar substance should be so placed as to hold the flap in position until healing has taken place.

CORNEA.

Anatomy. The cornea forms the anterior part of the fibrous coat of the eye. It is in form a horizontal ellipse, measuring 11 mm. in its vertical and 12 mm. in its horizontal meridians. At the periphery the cornea is 1 mm. in thickness, but at the centre it is slightly

thinner. The radius of curvature of the anterior surface of the cornea is variously estimated at 7.5 mm. to 8 mm. Since the radius of curvature of the scleral portion of the globe is 12 mm., it will be readily seen that the cornea is more sharply curved than the sclera. A slight annular depression is found at the anterior margin of the sclera—the union of the cornea with the sclera—known as the *sulcus*

FIG. 138.



Sectional view of cornea. (SCHAEFER.)

scleræ. Although oval in form anteriorly, posteriorly the cornea is circular. The sclera overlaps the corneal tissue externally, the overlapping being greatest above and below. The cornea is composed of five layers (Fig. 138):

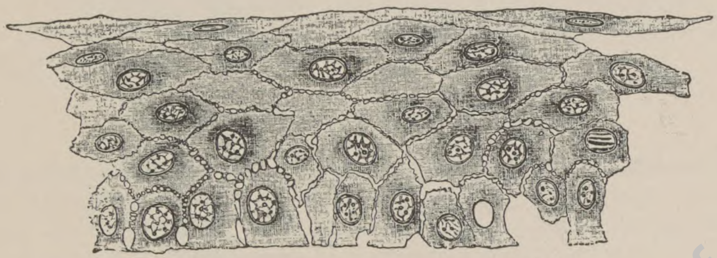
1. The epithelial layer, which is stratified; the superficial layer of cells is composed of tessellated or pavement epithelium. The cells

of the middle layer are irregularly cuboidal in form, and are supplied with numerous fine processes ("prickle cells") which interlace with the processes of the adjoining cells. The cells of the deep or basement layer are columnar or cylindrical in shape, are somewhat irregular in length, and are placed on a basement membrane. All of the cells of the cornea are supplied with nuclei. Regeneration of cells takes place from all the layers.

2. The second layer is a thin membrane, and is known as Bowman's membrane. (Fig. 139.)

3. The third layer is the thickest layer of the five, and is known as the *substantia propria.* It consists of numerous bundles of connective-tissue fibres associated in thin layers—lamellæ. The lamellæ are arranged parallel to the surface of the cornea. They are joined by connective-tissue fibres which pass from one lamella to another. The connecting fibres are so numerous in the anterior part of the substantia propria that they are given the name of *fibræ arcuatæ.*

FIG. 139.



Anterior epithelial layer of cornea. (QUAIN.)

Lying between the bundles and lamellæ are small spaces known as lacunæ, and, uniting these, numerous small canals known as canaliculi. These lacunæ and canaliculi together form the canalicular lymph system of Recklinghausen. In each lacuna a branching cell is found whose protoplasmic processes extend along the canaliculi, anastomosing with those of adjacent cells. These cells are known as the fixed cells of the cornea, in contradistinction to the leucocytes, which, by amœbic movements, penetrate to every part of the cornea, and are known as the migratory cells of the cornea.

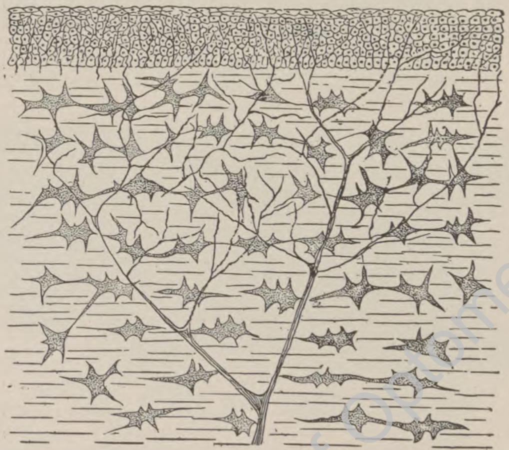
4. The fourth layer of the cornea is a thin homogeneous membrane, known as Descemet's membrane, which possesses chemical properties that serve to distinguish it from Bowman's membrane. Ranvier is of the opinion that this corneal membrane is the product of the endothelial cells of the cornea which rest upon it. The membrane of Descemet breaks up into numerous fibres at the periphery of the cornea, forming the ligamentum pectinatum.

5. This layer consists of a single layer of polygonal cells of the endothelial variety which lose their peculiarities at the ligamentum

pectinatum, passing over into cells that are much thinner and that cover the fibres of this ligament.

Nerves. These are derived from the ciliary plexus formed by the long and short ciliary nerves. (Fig. 140.) They pass through the sclera on the outer side of Schlemm's canal and form a network, the *plexus annularis,* in the vicinity of the margin of the cornea. From this plexus two sets of twigs are given off, one set passing to the conjunctiva, where they join the conjunctival nerves and form a plexus. From this plexus a number of nerve trunks are given off, which enter the cornea and supply the anterior layers of that structure. The second set of twigs pass directly to the substantia propria corneæ, entering it near Descemet's membrane.

FIG. 140.



Oblique section of the human cornea, showing ramification of the nerves. (DE WECKER.)

Radial fibres which leave the nerve trunks at the nodes of Ranvier pass to Bowman's membrane, which they pierce and form a plexus, the subepithelial plexus, from which terminal fibrillæ are derived, which end in the epithelial layer in nerve plates, peculiar convolutions, bulbs, hooks, and free ends.

Bloodvessels do not occur in the cornea, except at the limbus, where the episcleral bloodvessels end in a circle of looped capillaries.

Diseases of the Cornea.

Diseases of the cornea are included under the general term keratitis. They may be considered under two headings—suppurative and non-suppurative.

Histological Considerations. Regeneration of Corneal Tissue. It is at present conceded that the regeneration of corneal tissue proceeds from cells that migrate into the corneal tissue and form the mixed

cells of the cornea. When regression of a corneal ulcer has commenced, formative elements are found at the edge of the ulcer which gradually develop into connective-tissue fibres; this process continues until the defect in the cornea is filled up by the new tissue. The new fibres are not arranged in lamellæ, nor are they disposed parallel to the surface of the cornea. This irregularity of the disposition of the fibres causes a loss in transparency; the tissue so formed is opaque. In the healing of an ulcer the surface becomes covered with epithelium before the defect has been filled up by the deposition of the new fibres. The development of new-formed tissue continues until eventually the epithelial layer is raised to its normal height. In a certain proportion of cases complete filling of the defect does not take place. The area involved in the ulcerative process in these cases is often flat, forming what are known as *facets*. Bowman's membrane is never regenerated. Accompanying ulceration of the cornea, if the process is at all widespread, and in some cases where the tissue affected is not extensive (the invasion not deep), irritation of the iris is produced sufficient to bring about an exudation of lymphoid cells and of the coagulable portions of the blood into the anterior chamber. This exudate constitutes what is known as *hypopyon*.

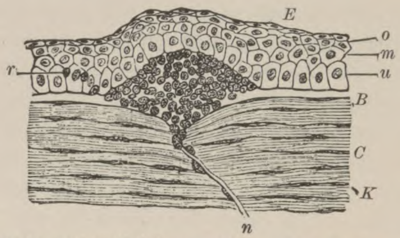
Suppurative Forms of Keratitis.

Eczematous Keratitis (Phlyctenular Keratitis; Scrofulous Keratitis; Lymphatic Keratitis). The etiology and pathology, except in regard to the affection of the tissues of the cornea, are the same as in eczematous conjunctivitis. The disease is met with most frequently in children between the ages of two and twelve years, but may appear in individuals up to the age of forty years. In many of the patients with eczematous (phlyctenular) keratitis obstructive rhinitis and adenoid tissue in the vault of the pharynx are found.

Symptoms. Severe irritation, as of a foreign body in the eye; pain of a neuralgic type, often extending to the orbit and temple; profuse lacrymation; photophobia, which is often intense, causing clonic blepharospasm in the milder cases and tonic blepharospasm in severe cases. In some cases the photophobia is so intense that the patient cannot be induced to open the eyes, even in a moderately lighted room, but avoids the light in every possible way. The intense photophobia is due to irritation of the terminal sensory nerve filaments, which are so richly supplied, to the corneal epithelium. (Fig. 141.) On forcibly separating the lids, there is often a gush of lacrymal fluid mixed with flakes of mucus. There is hyperæmia of the ocular conjunctiva, particularly in the vicinity of the phlyctenule. The vesicles may be single or multiple. They may form at the margin of the cornea only, may be distributed over the surface of the cornea, or may produce peculiar figures. The phlyctenule develops as in the conjunctiva, the apex softens and disappears,

and an ulcer results. The ulcer in many cases involves the superficial lamellæ of the cornea and leaves a faint opacity when it heals, due to the presence of a thin layer of cicatricial tissue.

FIG. 141.



Efflorescence on the cornea in conjunctivitis eczematosa. The nodule, which consists of cells, lies between Bowman's membrane, *B*, and the epithelium, *E*, which latter is thus raised so as to form a prominence. In the epithelium we distinguish the lowermost layer of cylindrical cells, *u*, the middle layer of polygonal cells, *m*, and the uppermost layer of flat cells, *o*; scattered between the epithelial cells lie a few round cells, *r*. A nerve, *n*, is seen extending through the parenchyma of the cornea, *C*, and among the corneal corpuscles, *K*, up to the nodule. (After IWANOFF.)

A peculiar form of eczematous keratitis is that known as *fascicular* or *frenular keratitis*. This is characterized by the formation of successive phlyctenulæ, another appearing directly in advance of a

FIG. 142.

Eczematous efflorescence in the limbus. The sclera, *S*, is distinguished by its more delicate fibrillation and its bloodvessels from the more homogeneous non-vascular cornea, *H*. The nodule is situated at a point corresponding to the boundary between the two membranes, but more over the sclera than over the cornea. It consists of densely packed round cells, between which the bloodvessels are recognizable under the form of lighter colored striæ. In the vicinity of the nodule the vessels of the conjunctiva, *c*, and episclera, *e*, are bordered by extravasated leucocytes. The epithelium, *E*, of the conjunctiva is bulged forward by the nodule, and at the apex of the latter is thinned and, owing to the penetration of the round cells into the epithelial layer itself, has lost the sharp border ordinarily existing between it and the connective tissue. Magnified 62 × 1. (FUCHS.)

subsiding one, the process beginning at the margin of the cornea. (Fig. 142.) The phlyctenule at the apex is connected with the conjunctiva by a leash of vessels which lie in the track of the disease.

The process often extends far onto the cornea, and forms a curved tract. On subsiding, an opaque stripe is left, which has been termed the *scrofulous band.*

Complications. A phlyctenular ulcer may extend, rapidly produce perforation of the cornea, and result in much damage to the eye.

Diagnosis. Eczematous keratitis may be confounded with herpes of the cornea, but the history of the case will suffice to differentiate between them.

Prognosis. Is favorable in nearly all cases.

Treatment. Local and constitutional treatment should be employed. It is often difficult to inspect the cornea on account of the photophobia and blepharospasm. If a drop of a solution of cocaine be instilled, the photophobia and blepharospasm will be much relieved. With many children it is necessary to place the head between the physician's knees before attempting to expose the cornea; a lid retractor is often required. The cornea is sometimes hidden under the upper lid, and it is necessary to wait, with the lid retracted, for it to come gradually into view. In severe cases a little ether or chloroform may be given.

The eye should be bathed or douched three or four times a day with a solution of boric acid. Atropine may be instilled sufficiently often to keep the pupil dilated. If the phlyctenule has broken down, calomel may be dusted onto the cornea once daily. An ointment of the yellow oxide of mercury (1 per cent. in vaseline) may be put into the eye twice daily. Fissure of the outer commissure often exists in these cases. The blepharospasm may be relieved to some extent by touching the fissure with a stick of nitrate of silver or with a crystal of the sulphate of copper (Koller). Obstructive rhinitis and postnasal growths should receive appropriate treatment.

Systemic treatment should be instituted as for eczema of the conjunctiva.

In fascicular keratitis the cure can be hastened by cutting the leash of vessels at the sclerocorneal margin by means of a sharp spud.

Ulcers of the Cornea. Ulcers of the cornea are variously classified. In regard to their development, they are primary, beginning in the cornea itself, or secondary, by extension of the process from the conjunctiva or from contiguous tissues.

In regard to position, they are marginal or central.

As to involvement of tissue, they are superficial or deep.

As to shape, they are circular, crescentic, punctate, dendritiform, filamentous, and irregular.

In character they are simple or infected. To the latter belong the so-called mycotic forms.

In regard to the stage of development, they are progressive or regressive.

Ulcers of the cornea present certain symptoms in common. (Fig. 143.)

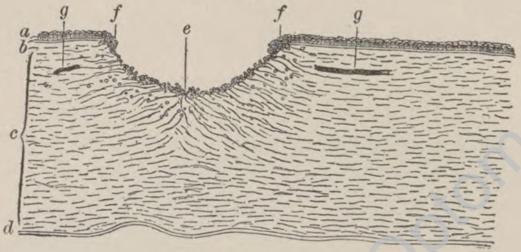
In all there are loss of corneal substance and more or less opacity of the cornea at the site of the ulcer and in its vicinity. Photophobia and pain are present in all but the neuroparalytic form. Impairment of vision occurs in all cases in which the pupillary area is involved. Pericorneal injection, partial or complete, with more or less secretion, is always present. The iris, ciliary body, and, in rare cases, the choroid, may become involved in the inflammatory process, and should receive appropriate attention.

Simple Ulcer. Simple ulcer of the cornea does not tend to advance. It may possess a grayish base, but often is clear and clean, and tends to heal rapidly. The ulcer may have any form, but is usually circular.

Cause. Simple ulcer is usually traumatic; but the term is also applied to those forms of infected ulcer in which the progress is speedily arrested.

Treatment. Simple cleanliness with, perhaps, the use of a boric acid solution or a solution of mercuric chloride (1 : 10,000) is all that is required.

FIG. 143.



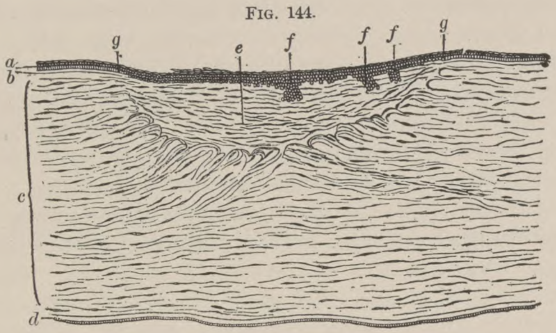
Corneal ulcer. (SAEMISCH.)

Infected Ulcer. The term infected ulcer is applied to ulcers which develop after solution of continuity of the corneal tissue, due to the entrance of a pyogenic micro-organism, such as the Staphylococcus pyogenes aureus, streptococcus, pneumococcus, etc. All infected ulcers are associated with an infiltration of the corneal tissue which extends to a greater or less degree from the margins of the ulcer. Infected ulcers may be marginal or central, circular or crescentic, or irregular in form, and pursue a course dependent on the nature of the micro-organism that has invaded the corneal tissue, on the location and on the ability of the corneal tissue to resist the destructive influence of the micro-organism. To this class belong some forms of crescentic keratitis, the so-called "serpent ulcer," "oyster shuckers' keratitis," and some of the forms of hypopyon keratitis.

Marginal Keratitis. This term is applied usually to the keratitis that accompanies eczema of the conjunctiva, which has been described under the heading of Eczematous Keratitis. Another form of marginal keratitis is the form that appears in the shape of a long

crescent involving from one-fifth to two-thirds of the circumference of the cornea, sometimes the entire circumference of the cornea, and to which the term "crescentic ulcer" sometimes is applied. This variety presents itself as an interrupted line of grayish infiltration immediately beneath the epithelium, occurring just beyond the free margin of the limbus. To the unaided eye, this seems to be a continuous line; but examined with the lens it is seen to be made up primarily of minute pustules which rapidly coalesce. Within twenty-four to thirty-six hours the epithelium covering these pustules disappears, and a superficial ulcer results. This process tends to advance toward the centre of the cornea regularly, the line of demarcation being quite clearly cut. With the advance of the superficial infiltration the epithelial cells disappear, but the deep layers of the cornea, save for a scanty infiltration of small cells, seldom are invaded. The affection, particularly in poorly nourished individuals, may advance until a large

FIG. 144.



Cicatrix of corneal ulcer. (SAEMISCH.)

part of the epithelium of the cornea has disappeared. The superficial lamellæ of the cornea are also sometimes affected to such an extent that when the ulcer has healed a delicate cicatrix, indicated by the presence of a thin opacity, remains. (Fig. 144.) This form of keratitis is usually monocular.

Cause. It is highly probable that a condition of the corneal tissue which renders it less capable of resisting the inroads of micro-organisms is an essential element in the development of this condition. Micro-organisms that have been observed in this form of ulcer are Staphylococcus pyogenes aureus, Klebs-Loeffler bacillus, and the pneumococcus.

Duration and Symptoms. This form of marginal keratitis seldom is met with in children, but is most frequent in adults of advanced years. It progresses slowly, is attended with pronounced symptoms of irritation, and is accompanied by more or less secretion from the conjunctiva. Injection of the palpebral as well as of the ocular conjunctiva is present. The process may terminate in a few days, but in some cases three months may elapse before recovery takes

place. Certain forms of marginal or crescentic keratitis bear a close resemblance to dendritiform keratitis.

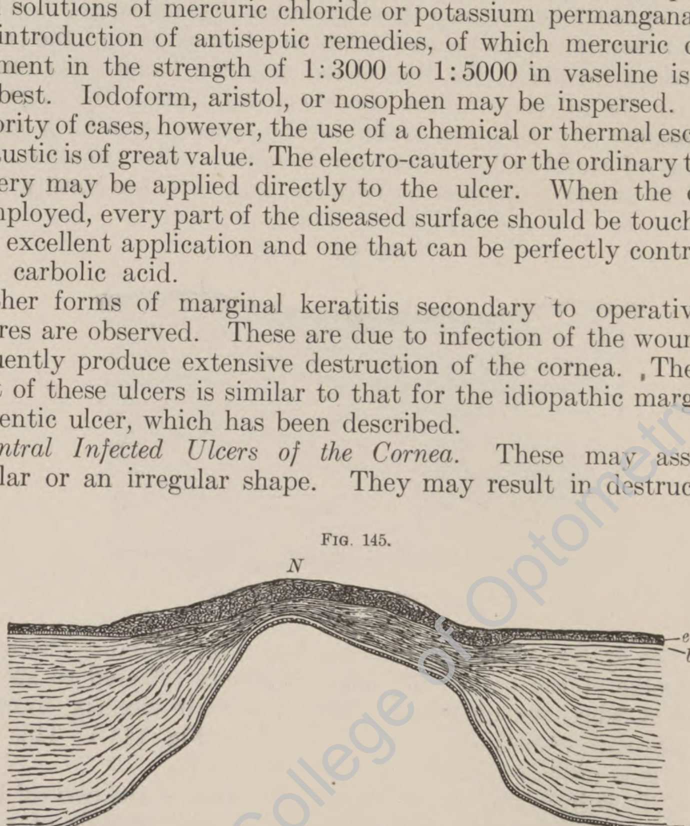
Diagnosis. The diagnosis of crescentic ulcer presents no difficulties, since the location and form are the essential features.

Treatment. In addition to the local treatment, the general health of the individual should be carefully investigated and measures adopted for improving it. Locally, treatment consists in cleansing the eye from time to time with a solution of boric acid, 3 per cent., mild solutions of mercuric chloride or potassium permanganate, and the introduction of antiseptic remedies, of which mercuric chloride ointment in the strength of 1:3000 to 1:5000 in vaseline is one of the best. Iodoform, aristol, or nosophen may be inspersed. In the majority of cases, however, the use of a chemical or thermal escharotic or caustic is of great value. The electro-cautery or the ordinary thermo-cautery may be applied directly to the ulcer. When the cautery is employed, every part of the diseased surface should be touched. A very excellent application and one that can be perfectly controlled is pure carbolic acid.

Other forms of marginal keratitis secondary to operative procedures are observed. These are due to infection of the wound, and frequently produce extensive destruction of the cornea. The treatment of these ulcers is similar to that for the idiopathic marginal or crescentic ulcer, which has been described.

Central Infected Ulcers of the Cornea. These may assume a circular or an irregular shape. They may result in destruction of

FIG. 145.



Keratectasia produced by an ulcer. The thinned and protruding cicatrix is distinguished by its denser texture from the adjacent normal cornea. The epithelium, *e*, over it is thickened, while Bowman's membrane, *b*, is wanting. On the other hand, Descemet's membrane, *d*, with its epithelium, is everywhere present, a proof that the ulcer has not perforated. Magnified 25 × 1. (FUCHS.)

a small or a large portion of the corneal tissue, or in destruction of the entire cornea. The form of ulcer to which the term circular is applied occurs most frequently in the shape of a cone whose apex is in the stroma of the cornea. The walls of the ulcer present a grayish appearance, and infiltration of the cornea extends quite regularly into the adjacent tissues. In some cases hernia of the anterior chamber results. (Fig. 145.) In some cases perforation takes

place, and in others plastic lymph forms at the bottom of the anterior chamber, when a condition known as hypopyon is produced. The circular ulcer is usually a relatively benign form, and regression sets in before perforation occurs. The ulcer when first noticed presents as a superficial loss of epithelium with a grayish-yellow centre. This grayish-yellow centre represents a mass of superficial necrotic tissue, which liquefies and extends slightly until within twenty-four or seventy-two hours a relatively deep loss of tissue results. This form of ulcer may occur at any period of life, but is most frequent in adults. Its duration is usually from five to ten days. In severe cases perforation, fistula, and partial staphyloma may result.

Treatment. The measures previously spoken of, comprised by the terms cleanliness and asepsis, should be employed. In many cases the inspersion of calomel once daily, with the introduction of the ointment of mercuric chloride four times a day, will suffice to bring about recovery; but in all cases of circular ulcer either the actual cautery or pure carbolic acid may be employed with very excellent results, and recovery brought about much more rapidly than by the use of simple medication. The condition of the system should always be inquired into, and suitable measures instituted to correct irregularities.

Hypopyon Keratitis. In all pronounced cases of corneal ulcer the anterior segment of the vascular coat of the eye (iris and ciliary body) is more or less irritated. As a result, exudation from the vessels ensues. The aqueous humor becomes more albuminous, and fibrin forms. Fibrin is deposited on the posterior surface of the cornea, and often on the other surfaces that are in contact with the aqueous humor. If the irritation is sufficient, leucocytes escape from the vessels of the iris and ciliary body, and reach the anterior chamber and gravitate to the bottom. If sufficient fibrin is present, the leucocytes become imbedded, forming a pultaceous mass which does not change its position when the position of the head is changed. In the absence of sufficient fibrin the collection remains fluid and changes its position as the position of the head is changed. The mass is yellow in color, and resembles pus. It contains no pathogenic germs and is innocuous. Its presence constitutes what is known as hypopyon. The superior border of the hypopyon is often slightly convex. On looking obliquely into the anterior chamber, it will be seen that the pus is chiefly deposited on the posterior surface of the cornea, the surface of the iris being free to a considerable distance below the upper margin of the deposit. The researches of Leber make it evident that the presence of the leucocytes and fibrin is an attempt on the part of nature to aid in preventing destruction of the corneal tissue. Many of the leucocytes penetrate into the corneal tissue by way of Fontana's spaces and proceed to the vicinity of the ulcer. The excess remain in the anterior chamber, and there form the collection just described. Older observers, noting the convex upper border of the mass of exudation in the anterior chamber, and the fact that

the iris was free, supposed that the collection of pus was between the lamellæ of the cornea, and, because of the resemblance to the lunula of the finger-nail, termed it *unguis* or *onyx* (nail).

The presence of hypopyon in itself calls for no particular treatment. With subsidence of the disease which calls it into existence it disappears by absorption, sometimes slowly, sometimes rapidly. It may disappear and reappear, and vary in amount from day to day.

Acute Sloughing Ulcer (*Serpent Ulcer*, Saemisch). This form of ulcer affects adults almost exclusively. It may occur in children debilitated by exhausting disease. Infection through a wound, usually superficial, is the cause, the pathogenic micro-organism being introduced either at the time of the traumatism, or subsequently, from the contents of the conjunctival sac. This affection has been observed frequently in farmers during the harvest season, and has been termed "harvesters' keratitis." Stonemasons are particularly liable. A similar form of corneal ulcer has been observed in those engaged in opening oysters, and has been termed "oyster shuckers' keratitis" (Rudolph). The micro-organisms chiefly engaged in the production of acute sloughing keratitis are the pneumococcus (Fraenkel, Weichselbaum, Sattler, Gaspanini, Pearls, Basso, Uhthoff, and Axenfeld), streptococcus, Staphylococcus pyogenes aureus, gonococcus, Klebs-Loeffler bacillus, etc. The term serpent ulcer is not descriptive of the disease, and should be abandoned.

According to Fuchs, the characteristic clinical picture of the disease is present only in the early stage. It then appears as a disk-like opacity near the centre of the cornea, the centre of the disk being not so dense as the margin. The surface, which at first is slightly raised, soon becomes slightly depressed. Iritis, cyclitis, and hypopyon develop early. The ulcer advances irregularly; an arrest may occur in one portion, and the epithelium may advance over the edge of the defect. At another part the gray infiltration advances, and loss of corneal tissue continues. Hernia of the anterior chamber, followed shortly by perforation, is the usual course. The entire cornea may become involved and slough. As complications, there may be loss of the crystalline lens, escape of vitreous humor, detachment of the retina, intra-ocular hemorrhage, panophthalmitis, etc.

Treatment. This should be energetic from the start. If the ulcer is small and in the early stage, medicinal treatment may be employed, provided the patient is under close observation and can devote the time to treatment. This consists in hot bathing, which should be continuous during the waking hours. Atropine, in 1 to 3 per cent. solution, should be instilled sufficiently often to keep the pupil dilated. Labarraque's solution, 1:10 to 1:8, may be instilled every two hours. Iodoform may be inspersed and a compress bandage applied. If treated as an "out" patient, operative procedures should be resorted to at once. In the early stage, if a large area is not involved and little hypopyon is present, cauterization should be resorted to, preferably with the actual cautery. Atropine should have been instilled

previously. After cauterizing, iodoform may be dusted into the wound and a compress bandage applied; or the conjunctival sac may be filled with bichloride vaseline 1:5000, and the eye bandaged. The eye should be inspected, the remedies used, and the bandage reapplied from one to three times in twenty-four hours. In ulcers that are large with large hypopyon the Saemisch incision should be performed. This is done by piercing the clear cornea at the margin of the ulcer, carrying the point of the knife into the anterior chamber beneath the ulcer, and making the counter-puncture in clear cornea. The incision is completed by cutting through the floor of the ulcer. The aqueous humor is permitted to escape slowly. The lips of the wound are separated, and the pultaceous mass forming the hypopyon is washed out or lifted out with a spatula. This operation permits of incarceration of the iris, but prolapse seldom occurs if the operation is performed with ordinary caution. The margins of the wound close rapidly. It is advisable to open the wound once every day for a few hours. Bandaging and the usual medicinal treatment should follow the operation.

Ulcer in Variola. In patients suffering from smallpox the cornea may be the site of one or more pustules. These do not, as a rule, lead to destruction of the eyeball, but they are followed by more or less opacity of the cornea, which greatly impairs vision. In France 35 per cent.—and after the introduction of vaccination 7 per cent.—of all the blind lost their vision from smallpox. In the United States, where vaccination is so general, blindness due to smallpox is seldom seen. If the eyes are inspected daily during the illness, and cleansing by means of mild antiseptic solutions and bland antiseptic oils or ointments is employed, something in the way of prevention may be accomplished. In addition to the formation of variolous pustules the cornea may be the site of a destructive keratitis, due to infection from other germs, as occurs in those who are not infected with variola. The results, on account of the depressed condition of the individual, may be very serious so far as vision is concerned.

Abscess of the Cornea. A process termed annular abscess of the cornea sometimes occurs after perforating wounds and after operations on the cornea which result in infection of the cornea. In these cases a yellowish annular ring forms in the central portion of the cornea. The centre of the cornea and the zone next the limbus are hazy, but less densely so. The yellow ring extends, involving the entire cornea. The corneal tissue sloughs. Destruction of the cornea in these cases is complete in three or four days. Destruction of the eye is sure to result. Fuchs advises enucleation at an early stage.

Fistula of the cornea appears as a small black point at or near the centre of the cornea. If perforation, the result of ulceration, occurs in the centre of the cornea, it may not be covered by the iris, and recovery must take place by the development of new connective tissue from the margins of the ulcer. Recovery under these con-

ditions is slow, and it sometimes happens that with closure of the perforation the intra-ocular tension is sufficient to rupture the delicate new-formed tissue, and the anterior chamber is evacuated and a fistula is formed. If the fistula remains open, the eyeball gradually atrophies or infection occurs, and panophthalmitis follows. The fistula may close, and on restoration of the intra-ocular tension may again be opened. This may be repeated a number of times. Infection usually occurs sooner or later, and loss of the eye results.

Treatment. In recent cases an attempt may be made to secure closure by applying a compress bandage, and myotics may be employed to keep the intra-ocular tension reduced. If the anterior chamber becomes restored, an iridectomy may be performed for the same purpose. Cauterization of the walls of the fistula is resorted to sometimes for the production of a firm scar; but it should be employed with care, to avoid wounding the lens. Excision of the walls of the fistula may be resorted to, and the opening may be closed by a suture.

Filamentous Keratitis. This disease may be classified as idiopathic or traumatic.

a. Idiopathic filamentous keratitis is characterized by the formation in the corneal epithelium of epithelial globules, measuring usually 1 mm. to 1½ mm. in diameter, which are pushed above the level of the epithelium, and finally become pendent from the epithelial surface and connected by small pedicles (Nuel, Hess). The globule consists of epithelial cells, of the tessellated variety, which are undergoing mucoid degeneration. In the centre a hyaline body usually is observed, resembling coccidium. The pedicle consists of a central core, composed of epithelial cells which have become elongated until they assume the appearance of fibrillæ. These are twisted into a small thread. The masses rise and fall, accompanying the movements of the lids. The filaments last from three to four days. A fresh group of the globules may appear. The number of globules may be but two or three, or they may be numerous and cover almost the entire cornea.

This disease is peculiar to advanced adult life, and is observed more frequently in eyes that have been the seat of an inflammatory process affecting the anterior segment. Similar globules may form from the floors of ulcers that are in the process of healing.

b. Traumatic filamentous keratitis is probably due to the adherence of partly detached threads of epithelium rather than to filaments derived from the eruption of globules as above described.

Symptoms. The development of filamentous keratitis is accompanied by symptoms of irritation, slight injection of the ocular conjunctiva, a sensation as of a foreign body in the eye, and the presence of scanty mucoid or mucopurulent secretion, portions of which adhere to the corneal surface. Slight febrile reaction sometimes is observed.

Treatment. Treatment should be constitutional as well as local. Local treatment consists in keeping the cornea cleansed, using mild

antiseptic solutions—solution of boric acid (3 per cent.) or potassium permanganate (0.2 to 1 per cent.) may be employed. In addition, much comfort is given the patient if a lubricant in the form of borated (5 per cent.) or bichloride vaseline (1:5000) is introduced into the eye three or four times daily.

The systemic treatment consists in the employment of general tonic remedies. Under this form of treatment we may hope for a subsidence of the affection in from three to five days. Recurrence may take place.

Dendritic Keratitis (*Furrow Keratitis; Mycotic Keratitis; Keratitis Arborescens*). The term is applied to a superficial form of keratitis supposed to be due to the presence of a specific micro-organism. The process usually begins at the periphery of the cornea, but may also begin in the centre of the cornea. It is characterized by the appearance of a narrow grayish line of infiltration in the corneal tissue near the surface, accompanied by an elevation of the epithelium. The epithelium covering this line of infiltration breaks down, and a shallow groove ("furrow") is formed. Preceding or following the destruction of the epithelium, offshoots from the original line of infiltration are observed. The offshoot frequently terminates in a minute grayish enlargement (colony). These offshoots multiply, until eventually a tree-like ("arborescent") formation is presented. The infiltration is not confined to the grooves and lines, but advances into the surrounding tissue for a short distance as the process continues. The advance of the process differs in different cases. As a rule, the advance of the disease is relatively slow, a number of days, perhaps weeks, elapsing before full development is reached. The superficial nature of the process may be continued throughout its course. Involvement of the deeper tissues may supervene, and in exceptional cases perforation of the cornea may take place. When the deeper tissues are involved, it is the result of a mixed infection of the cornea.

Symptoms. The symptoms of this affection are frequently very distressing: the patient complains of a sensation as of a foreign body in the eye; photophobia to a very annoying degree develops. Neuralgic pains, affecting the distribution of the supra-orbital and infra-orbital branches of the fifth nerve are experienced. These symptoms may suddenly cease and be absent for a day or two, and then suddenly recur. The intensity of the symptoms depends directly upon the activity of the process.

Cause. A cause has not as yet been determined. The appearances indicate the presence of a micro-organism.

Desiccation Keratitis (*Keratitise Lagophthalmo*). When the cornea is exposed for any length of time without suitable lubrication, the epithelial cells become dry and irregular and the cornea becomes slightly opaque. Exposure leads to one of two things: the corneal tissue either takes on a cutaneous condition, or the corneal substance is lost, and ulcer occurs. Keratitis from desiccation occurs

in cases of extreme exophthalmos, retraction of the levator palpebræ superioris, extreme ectropion, paralysis of the orbicularis palpebrarum preventing closure of the eyes; in debilitating diseases, as typhoid fever, smallpox, and in the later stages of disease ending in the death of the patient, where, from inability to close the eyes, the cornea is exposed. In all cases of desiccation keratitis the portion of cornea first affected is the exposed portion.

Desiccation keratitis is probably most frequently observed in lagophthalmos. The ulcer may progress and destroy more or less of the cornea.

Symptoms. The symptoms are, as a rule, not severe. More or less pain referable to the eye is complained of, and in cases of involvement of the iris and ciliary body, symptoms peculiar to disease of these structures develop.

Treatment. The treatment consists in providing protection for the exposed cornea. This, in the cases of lagophthalmos and exophthalmos is accomplished by the application of protective bandages and the use of lubricating ointments. In cases not admitting of relief by spontaneous recovery, tarsorrhaphy to an extent sufficient for the protection of the cornea may be performed. After cicatricial ectropion plastic operations for restoring the lids should be resorted to.

Keratomalacia. Keratomalacia accompanies xerosis epithelialis, affecting infants, and occurs in the later stages of exhausting diseases, such as typhoid fever, scorbutus, etc. It is characterized by a grayish discoloration of the cornea, increase in thickness of the corneal tissue, accompanied by softening and complete loss by sloughing. Keratomalacia is seldom seen in adults. The condition belongs almost entirely to xerosis affecting infants, as has been stated in the description of xerosis (see Conjunctiva), and needs no further reference here. A lethal result almost invariably takes place in those affected.

Treatment manifestly is of no avail.

Neuroparalytic Keratitis. Diseases of the fifth nerve occurring either in the trunk of the nerve, in the Gasserian ganglion, or at the nucleus of the nerve, produces insensibility of the cornea, and at the same time removes the source of stimulation for the lacrymal gland, causing a diminution in the secretion of the gland. With loss of the sensibility of the cornea and conjunctiva the individual fails to appreciate the presence of foreign substances, and is not made aware of atmospheric influences on the surface of the cornea, which undergoes desiccation. Winking is not performed sufficiently frequently to maintain the proper moisture of the cornea or to remove foreign substances from its surface. This condition leads to partial loss of epithelium and permits of the entrance of destructive bacteria, bringing about a keratitis which at first is superficial and general, but later may develop into deep ulceration with loss of substance, perforation, and, in the severer cases, complete loss of vision. It is held by some writers that this process is not entirely due to exposure,

but that trophic disturbances occur consequent on the destruction of the nerve. Since, however, it is not proved that trophic nerves, so-called, exist, we are not warranted in assuming that they do exist, and we must attribute the changes that occur in the cornea to insensibility of the cornea, to exposure, and to the entrance of noxious germs.

Cases are cited by a number of writers in which, after protection to the cornea has been furnished, the process has not abated, but has continued, and destruction of the cornea has resulted. These observations, however, were made prior to a perfect understanding of asepsis, and it is possible that destructive micro-organisms were present, and that the continuation of the diseased process was due to them.

Treatment. It is found that protection of the cornea either by means of a protective bandage, tarsorrhaphy, or by the use of ointments sufficiently frequently applied to keep the cornea covered, will bring about a restoration of the corneal tissue, and will prevent further development of the keratitis.

Non-suppurative Forms of Keratitis.

Pannus. This is a form of superficial vascular keratitis, and is the result of an attempt on the part of nature to protect the cornea from irritating influences. It is observed most frequently in trachoma, and occurs in that stage of trachoma in which the elevations on the surface of the conjunctiva are hard and dense, and are capable of producing displacement of the epithelium of the cornea with which they come in contact. The vascularity may affect the whole or a part, usually the upper portion, of the cornea. In cases of trachoma in which the lower lid is but slightly involved, the pannus of the cornea may be limited exclusively to the upper half. It sometimes occurs that the irritation of the corneal tissue affects only the lower half of the cornea, in which case the pannus is limited to this part. Vascular pannus may be extremely slight (*pannus tenuis*), or the vascularity may be very pronounced (*pannus vascularis*); it may be so intense that the cornea is converted into a condition resembling a fleshy mass (*pannus crassus* or *carnosus*). The vascularity remains as long as the irritation is present, and then gradually subsides, often leaving but few traces. In severe cases complete opacification of the cornea may result. In the later stages, when few bloodvessels are present, the condition is known as *pannus siccus*.

Pathology. The bloodvessels in pannus, in the earlier stages and lighter forms, are found immediately beneath the epithelial layer. They lie in a scant stroma of new-formed connective tissue or formative cells, and are accompanied by a more or less plentiful small-cell infiltration. In pannus tenuis Bowman's membrane remains almost, if not quite, intact. In the severer forms of pannus the superficial lamellæ of the substantia propria are involved and Bowman's mem-

brane is greatly changed, losing its character entirely. It is never restored.

Treatment. This consists in removing the cause.

Herpes Corneæ. This disease, which resembles eczema corneæ in some respects, is to be differentiated from it. It occurs as an accompaniment of herpes febrilis most frequently, but also accompanies herpes frontalis. It is characterized by the appearance in the cornea of two or more vesicles, which at first are transparent, but soon become cloudy and of a yellowish color. The epithelium then gives way, and there is a superficial ulcer with shreds of epithelium hanging from it. The floor of the ulcer is anæsthetic, but the surrounding cornea is not. The appearance of the vesicles is preceded by sharp, pricking pain, by lacrymation, and, at times, by mucopurulent secretion. The gray infiltration at the base of the vesicle may not extend, but fine lines of infiltration extending into the surrounding cornea may be present. Crops of vesicles are apt to occur.

This form of keratitis attacks adults of middle life most frequently. Younger individuals may be attacked. Accompanying herpes of the cornea there may be herpetic vesicles on the lips, nose, face, and eyelids.

Horner, who described this affection in 1871, observed it following pertussis, intermittent and typhus fever. According to Haab, the outer layers of the cornea, Bowman's membrane, and the epithelial layer may be elevated by the process. In cases that are suitably treated, recovery occurs as a rule in a week or ten days. In neglected cases secondary infection may occur, accompanied by more or less destruction of the cornea, hypopyon, and iritis.

Treatment should be local and constitutional. Cleansing the eye with a 3 per cent. solution of boric acid three or four times daily, and the introduction of bichloride vaseline (1:5000) after each bathing, will be sufficient.

Constitutional treatment should be directed to improvement of the general system.

Keratitis punctata superficialis begins with symptoms of acute conjunctivitis; it is related to herpes febrilis corneæ, but does not form vesicles. Small gray spots form in the superficial layer of the cornea, occupying the central portion; these may be very numerous, or may be limited to six or eight. The corneal surface is rendered uneven by the elevation of the epithelium lying over the spots of infiltration.

Bullous Keratitis. This condition consists in the formation of a large vesicle or vesicles on the cornea, usually occupying the lower portion of the cornea. It occurs in eyes that are affected by a chronic iridocyclitis, after superficial traumatism, and in those which are subject to increase of tension. The presence of the bleb or bulla is accompanied by symptoms of irritation. There are excessive lacrymation, photophobia, and mucopurulent secretion. The affection is peculiar to adult life.

Duration. The bleb usually persists for a few days, then ruptures, the threads of epithelium hanging from the margins; the denuded surface is rapidly recovered by epithelium. In some cases the bleb opens spontaneously at the superior part and the bleb wall becomes reattached to the cornea. Recurrences are frequent, the recurrence taking place in some cases within a few days, and in some cases months or years afterward.

Pathology. The outer wall of the bleb consists of the entire epithelial layer of the cornea, which has become raised by the transudation of fluid through Bowman's membrane. Brugger[1] believes that the first step is an infiltration of the substantia propria of the cornea with fluid which could not escape by the limbus; new tissue-elements form beneath the epithelium of Bowman's membrane, and the epithelial layer eventually becomes detached and is pushed forward.

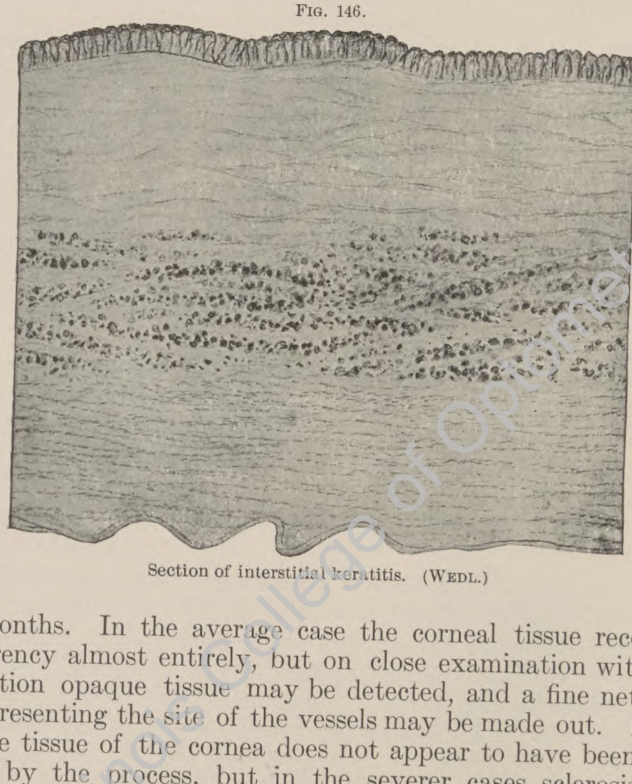
Treatment. It is sometimes sufficient to puncture the vesicle and apply a compress bandage. It becomes necessary in some cases to remove the anterior wall of the vesicle, and to treat the denuded surface by an application of a solution of nitrate of silver 0.5 per cent. to 1 per cent., or by superficial cauterization. In some cases removal of the superficial parts of the cornea has been resorted to. It is, of course, desirable to cure the condition which makes the formation of the bleb possible. The ultimate result in the vast majority of cases is favorable for retention of the eye and the preservation of some degree of vision. In rare cases removal of the globe becomes necessary.

Parenchymatous Keratitis (Interstitial Keratitis; Diffuse Keratitis). Parenchymatous keratitis may develop from the periphery of the cornea or first manifest itself by the appearance of opacities on or near the centre of the cornea. When developing from the periphery, its free border is irregular and is usually less dense than at the margin of the cornea. If carefully examined, the infiltration will be found to be uneven in density, frequently being made up of a number of foci. Thickening of the conjunctiva at the sclerocorneal junction accompanies the process, the limbus apparently advancing a short distance onto the cornea. The limbus here is deeply congested and presents a bright-red border. The extension of the vessels of the conjunctiva onto the cornea is limited by a sharp line of demarcation, and seldom exceeds 1 mm. or $1\frac{1}{2}$ mm. Soon vessels make their appearance in the parenchyma of the cornea and extend into the infiltrated area. The infiltration advances toward the centre, and may eventually involve the whole of the cornea. The opacification may become limited, affecting only a small portion of the cornea. It seldom occurs that infiltration of the cornea advancing from the margin involves the entire margin of the cornea uniformly. In the majority of cases infiltration begins in the lower nasal quadrant.

[1] Monat. f. Aug. Heilk., 1886, vol. xxiv. p. 500.

The second mode of onset is that in which the opacity first manifests itself in the centre of the cornea. In these cases the infiltration occurs in numerous foci in the centre of the cornea, gradually spreading until the greater part of the cornea is involved. (Fig. 146.) The vessels of the limbus throughout the whole periphery of the cornea are somewhat injected, but the principal vascularity occurs in the deep tissues of the cornea, advancing from the periphery. In both forms of onset the surface of the cornea becomes somewhat irregular, due to numerous minute elevations. Vision is impaired in proportion to the density of the infiltration. The infiltration of the cornea advances rapidly, in some cases involving the entire corneal tissue in from two to four weeks. In some cases the advance is much less rapid, the height of the affection being reached only after two or

FIG. 146.



Section of interstitial keratitis. (WEDL.)

three months. In the average case the corneal tissue recovers its transparency almost entirely, but on close examination with bright illumination opaque tissue may be detected, and a fine network of lines representing the site of the vessels may be made out. In many cases the tissue of the cornea does not appear to have been greatly affected by the process, but in the severer cases sclerosis of the tissue results, the cornea becoming thinned, and its diameter increased by stretching due to the intra-ocular pressure. In some cases only a portion of the cornea becomes sclerosed and ectatic.

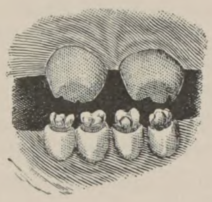
Duration. In the average case the disease runs its course in from five to ten months—seldom less than five months even in the mild cases. In the severer cases the clearing up of the cornea does not reach the maximum in less than two or three years.

Degree of Severity. Parenchymatous keratitis may affect only a small portion of the cornea at the periphery. It may occur in only one or two small punctate patches near the centre of the cornea. The opacities advance for a few weeks extremely slowly, and subside, leaving scarcely a trace. Severe forms may cause complete opacification of the cornea, reducing vision to perception of light. Parenchymatous keratitis is accompanied not infrequently with involvement of the sclera and of the anterior portion of the vascular membrane of the eye—iris, ciliary body, and choroid.

Symptoms. More or less irritation, increased lacrymation, photophobia in proportion to the rapidity of advancement of the disease, pain, sometimes extremely light in degree, sometimes quite severe, referable to the eye and to the temple, are experienced.

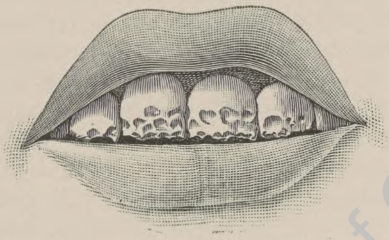
Cause. Parenchymatous keratitis is due in the vast majority of cases to syphilis, either inherited or acquired. Inherited syphilis is by far the most frequent cause. Rheumatism and gout are causes, perhaps in very few cases, and indefinite dyscrasia may also be said to produce a very small proportion of the cases. As a result of

FIG. 147.



Permanent incisors of inherited syphilis.

FIG. 148.



Rachitic teeth.

inherited syphilis, it may affect individuals between the ages of five and forty-five years. It occurs most frequently between the ages of seven and fifteen years.

Accompanying parenchymatous keratitis due to inherited syphilis are certain peculiarities in the conformation of the face and head. The forehead and cranium are larger in proportion to the lower part of the face, evidences of hydrocephalus being present in some individuals. The skin of the face has an old or parchment-like appearance; the skin about the mouth is wrinkled; small scars are observable at the angles of the mouth and also in the lips near the angles of the mouth, indicating the presence at some period of life of fissures of the lips. The inferior and superior maxillary bones are often narrower than normal, the superior maxillary being acutely arched.

Dentition in nearly all of these cases is of a peculiar type, affecting principally the upper incisors. (Fig. 147.) The teeth are not placed close together. They are usually smaller than normal, are broader at the base than at the apex, are peg-shaped, and present a notching of the free border. This form of dentition was described

first by Hutchinson as accompanying this affection, and is known as Hutchinson teeth. (Fig. 147.) It should not be confounded with that in which the enamel is defective at the apex of the teeth, or in which by a terraced appearance of the enamel different phases in its development are represented. Teeth of this character are usually the result of rickets and of other forms of malnutrition in the infant. (Fig. 148.) Not infrequently the hearing in infants with parenchymatous keratitis is defective, due to involvement of the auditory nerve.

In parenchymatous keratitis the result of acquired syphilis involvement of the cornea occurs from six months to as many years after the appearance of the primary sore.

Treatment. The treatment should be local and constitutional. In parenchymatous keratitis due to inherited syphilis the treatment is not the same as that employed in the cases due to acquired syphilis. In the first form the local treatment consists in the use of atropine for the purpose of maintaining dilatation of the pupil; the use, ordinarily, of stimulating applications to induce vascularization of the cornea; and modification of the light by dark glasses, suitable shades, or by keeping the patient in a dark room. In many cases of this disease stimulation is not necessary in the early stage, but stimulation, although not necessary in many cases, is never harmful, and, in the opinion of the writer, is desirable even in those cases in which the onset is relatively rapid. Stimulation is effected by means of the introduction of suitable ointments, either ointment of yellow oxide of mercury (1 to 1.5 per cent.) or mercuric chloride (1:5000), or the instillation of stimulating drugs, as the wine of opium, or by hot applications to the eye, usually by means of moist heat (hot bathing), either with salt water or a hot solution of boric acid three or four times a day, fifteen to thirty minutes each time). Powders may be dusted into the eye, the best being calomel. Calomel, when employed, should be introduced once a day.

Internal treatment consists in the general tonic treatment of the individual, and the administration of a mercurial with, possibly, potassium iodide. Mercurials have apparently a better effect than the iodide ; the mercurial may be administered satisfactorily in the form of calomel, 0.1 grain four times daily. Children bear medication of this kind even better than adults.

Parenchymatous keratitis due to acquired syphilis ordinarily does not require stimulation. In other respects the local treatment is similar to that employed in the inherited form. The constitutional treatment is the same as that ordinarily employed in acquired syphilis. It should be rigorously pushed.

Sclerosing Keratitis (von Graefe). This form of keratitis accompanying scleritis is, in fact, an extension of the process into the cornea. The opacity is of a grayish-white color and involves the deeper layers of the cornea. It advances slowly, seldom reaching the pupillary area. The opacification of the cornea does not extend

beyond the affection of the sclera at the periphery of the cornea; but if the scleritis entirely encircles the cornea, the sclerosis may have the same extent, and may eventually produce cloudiness of the entire cornea. The density of the opacity is least in the parts farthest removed from the margin of the cornea; the density may vary in the different parts of the sclerosed portion. On subsidence of the scleritis the opacification of the cornea subsides to a certain degree, the clearing up of the opacity being most marked in the portions farthest removed from the margin of the cornea. It seldom occurs that the opacification disappears entirely.

The opacity of the margin may become so slight that it can be detected only by oblique illumination. During these processes the corneal tissue is at no time thickened; as the opacification disappears the corneal tissue may become reduced in thickness. The surface of the cornea during these processes usually remains smooth; but in not a few cases the epithelium at the margin of the cornea becomes irregular, and some defects in it may occur.

Symptoms. The symptoms are those referable to the scleritis, and will be described under that heading.

Treatment. The treatment is the same as that employed in the treatment of the primary affection—the scleritis.

Striated Opacities of the Cornea. After operations necessitating incision of the corneal tissue, as a result of tight bandaging, and, in some cases accompanying ulceration of the cornea, long, narrow, grayish stripes are observed which traverse the cornea in various directions. They are apparently unaccompanied by inflammation. Following incisions of the cornea, the stripes commence apparently at the margin of the incision and extend in radiating lines to a considerable distance from the wound, sometimes traversing the entire cornea. They appear in from twenty-four to forty-eight hours after the incision is made, and disappear very gradually. Cases have been recorded in which the grayish lines remained permanently. The linear opacification may be so slight as not to interfere with vision in any way, and may be sufficiently dense to cause a decided diminution in vision. In striated opacity of the cornea resulting from tight bandaging, the lines may cross each other in all directions. They are usually most marked near the centre of the cornea. In striated opacification accompanying corneal ulcer, the striæ radiate from the margins of the ulcer. Knies, Fuchs, and others attribute this form of striated opacification to wrinkling of Descemet's membrane, that occurring after incision of the cornea being due to relaxation of the tension on the membrane at the point of incision, that from bandage pressure to a reduction in the tension of the globe or flattening of the cornea. Becker and von Recklinghausen demonstrated the fact that the lymph channels in the cornea are sometimes distended and their contents turbid. These lymph channels or tubes extend between the lamellæ of the cornea in straight lines. It has been thought that the channels through

which the nerve trunks pass become filled with turbid fluid and form striations in some cases.

Treatment. There is little to do for this condition. Resolution takes place in the majority of cases, and the transparency of the cornea is restored.

Infiltration of the Cornea Originating from the Posterior Surface. In cases where an exudation, a lens dislocated into the anterior chamber, or a cyst of the iris lies against the cornea, an opacity may form which may involve the entire thickness of the cornea. The cornea appears to be macerated at the point of contact, the surface being slightly elevated above that of the surrounding cornea. Even after removal of the cause the opacity persists, but the swelling disappears.

Deep Vascular Keratitis. This disease of the cornea develops by the advancement of minute vessels from the margin into the deep layers of the parenchyma of the cornea. It appears as a uniform pale-red or salmon-colored patch occupying a small portion of the margin of the cornea; it gradually broadens and extends into the corneal tissue, seldom passing beyond the pupillary margin, and not involving more than one-quarter of the circumference of the cornea. A very narrow margin of infiltration precedes the vascular patch. The patch is densest at the margin of the cornea, gradually becoming thinner until it ceases abruptly at its free border.

Symptoms. The disease is accompanied by symptoms of irritation, slight disturbances of vision, and photophobia. The ocular conjunctiva is congested in the vicinity of the vascularization of the cornea.

Cause. Deep vascularization of the cornea is due to either inherited or acquired syphilis. It is probably met with more frequently in acquired than in inherited syphilis.

Duration. The condition advances very slowly, and if not interfered with by treatment may last from three to six months. The cornea again becomes transparent with the exception of a very slight cloudiness. Keratectasia may eventually follow.

Treatment. Treatment should be local and systemic. Local treatment consists in stimulating the process by bathing with hot solutions (solution of boric acid, 3 per cent., being usually employed), the use of atropine to prevent posterior synechiæ in case of involvement of the iris, and the frequent introduction of some ointment, as the yellow oxide of mercury, 1 per cent., or the bichloride, 1: 5000, into the conjunctival sac. The systemic treatment should be the ordinary antisyphilitic treatment, employed vigorously for the first few weeks, and continued in a moderate way for some months subsequently.

Ribbon Keratitis (Trophic Keratitis; Bandolet Keratitis). This is a peculiar form of degeneration of the anterior layers of the cornea. It occurs in eyes that have been lost through glaucoma and in eyes affected by inflammation of the anterior segment of the globe, with more or less degeneration of the cornea. The epithelium of that

portion of the cornea corresponding to the opening of the palpebral fissure becomes slightly thickened and roughened, the cornea throughout this space becomes cloudy, and whitish plaques resembling deposits of calcareous material appear. The process develops extremely slowly, and is accompanied by symptoms of irritation of a very mild type. Vision is interfered with because of the formation of the opaque strip over the pupillary area.

Treatment. Aside from the formation of an artificial pupil for visual purposes, treatment is of no avail.

Non-inflammatory Conditions.

Arcus Senilis Corneæ (Gerontoxon). This occurs as a narrow grayish-white band which appears at the margin of the cornea. The band is separated from the limbus by a narrow strip of perfectly clear cornea; the line of transition is sharply cut; toward the centre of the cornea the gray band fades gradually into transparent cornea. This gray band is due to the presence of minute globules of fat, hyaline masses, and sometimes calcareous granules in the superficial layers of the corneæ. The change, which is peculiar to adults, depends on senile partial atrophy of the vessels at the limbus.

Treatment is not necessary.

Ectasiæ. The classification adopted by Fuchs is a very excellent one. He divides ectasiæ into those of inflammatory origin, which include *staphyloma* and *keratectasiæ;* and those of non-inflammatory origin, which include *keratoconus* and *keratoglobus.*

Staphyloma may be either partial, total, or multiple. Staphyloma is a protuberant cicatrix, the result of a perforating ulcer of the cornea, with involvement of the iris. The iris may be simply incarcerated, but it is usually primarily prolapsed. After a perforating ulcer of the cornea the cicatrix that forms may bulge as healing progresses, in which case it is termed *primary staphyloma;* or the cicatrix may be flat and bulge subsequently, termed in this case *secondary staphyloma.* The shape of the staphyloma is usually conical, particularly in the partial staphylomata; in total staphyloma it may be spherical, often the edges are abrupt, and they may even overhang the cornea. The degree of the protrusion varies greatly. In certain staphylomata cicatricial bands develop across the surface, extending in various directions, producing a lobulated condition known as *racemose staphyloma.*

Staphyloma may develop either because of the protrusion of the cicatrix due to the normal tension of the globe, the tissue being too weak to withstand the pressure, or, as is most frequently the case, the staphyloma develops because of an increase of intra-ocular tension —a condition of secondary glaucoma. In cases where the entire pupillary margin of the iris is involved in the cicatrix, the communication between the anterior and posterior chambers is shut off, and an increase in tension naturally follows, because of a closure of the nat-

ural ways for the escape of secretion from the interior of the eye. The increase in tension usually comes on extremely slowly. In rare cases, however, the increase in tension is rapid, and may be attended by inflammatory symptoms and by pain.

Consequences. In the early stages of the formation of staphyloma vision is interfered with in proportion to the extent of opacification of the cornea and the involvement of the free margin of the iris. Unless secondary glaucoma supervenes, perception of light is maintained because of retention of the integrity of the deeper tissues of the globe. With increase in tension the staphyloma continues to enlarge, and the retina, choroid, and ciliary body become atrophic. In many cases the transparency of the lens is lost, the lens shrinks, and in some cases is transformed into a thin opaque disk. In large staphylomata the apex is frequently exposed, and becomes ulcerated or takes on a cutaneous condition. Ectropion of the lower lid sometimes results.

The anatomical conditions present are as follows: The iris always lines the posterior surface of the protruding portion; superimposed on the layer of iris is a layer of cicatricial tissue, plus the elements of the cornea that have not been destroyed by the ulcerative process; covering this is a layer of epithelium. The epithelial layer is thickened and irregular. The thickness of the cicatrix varies in different parts. At the apex of the staphyloma no corneal tissue is found, but at the sides corneal tissue is present. In total staphyloma of the spherical variety it frequently happens that no corneal tissue enters into the formation of the protruding portion. The walls of the staphyloma may be extremely thin, perhaps one-third of the thickness of the normal cornea, or they may be thick and dense and contain calcareous deposits. Accompanying the protrusion of the cicatrix of the cornea we may also have a general enlargement of the globe.

Treatment. Much may be done to prevent the formation of staphyloma in partial anterior synechiæ, following ulceration of the cornea. During the process of healing of perforating ulcer of the cornea a compress bandage should be retained until a firm, flat cicatrix is formed. The eye should be examined from time to time, and if there is evidence of increased tension, or evidence of bulging of the cicatrix, a broad iridectomy should be made. After a staphyloma has formed, if it is partial and the anterior chamber has not become entirely obliterated, excision of a small crescent-shaped piece of the staphyloma may be practised in connection with the iridectomy. The eye may then be bandaged, and the bandage retained until a firm cicatrix has formed. It may be necessary to remove the lens in some of the cases treated in this manner. Ablation of the apex of the staphyloma may be practised in partial and in total staphylomata, the lens being removed at the same time. The gap thus produced may be permitted to close under a compress bandage, or it may be closed by sutures. In extensive staphyloma a very excellent plan is to close the opening by means of the conjunctiva, with or

without a primary closure with catgut sutures. In closure by means of the conjunctiva, the conjunctiva is dissected away from the globe, beginning at the limbus, and then, by either a continuous (tobacco pouch) suture or by interrupted sutures, may be drawn over the wound and the margins brought together.

In not a few cases of total staphyloma, ablation of the staphyloma simply, does not suffice. The protruding portion may be excised, the contents of the globe removed, and a glass or metal ball inserted. Removal of the contents of the globe (exenteration) or enucleation must be resorted to at times.

Partial staphyloma of the cornea is self-limited in a large number of cases. In total staphyloma the enlargement of the protrusion progresses. Superficial ulceration not infrequently occurs, producing some injection of the ocular conjunctiva, but seldom occasioning much pain. Perforation at the apex of the staphyloma occurs spontaneously; following this the opening closes, the staphyloma becomes flattened to a certain extent, the tension then gradually increases, bulging becomes marked, and rupture again takes place. This may be repeated a number of times; infection followed by panophthalmitis is apt to occur.

Keratectasia. By this term is meant a bulging of the cornea as a result of inflammation without perforation and without involvement of the iris. This condition may follow ulceration of the cornea, and softening of the cornea as with pannus. An opacity is always present at the apex of the protrusion. Keratectasia is almost always partial.

This condition should not be confounded with keratoconus, keratoglobus, or keratocele. Keratocele is a condition due to bulging of Descemet's membrane, the result of an ulcerative process that has destroyed the anterior layers of the cornea. It presents as a transparent bead-like bulging of Descemet's membrane in the centre of a grayish area—the infiltrated margins of the ulcer. Keratectasia may follow keratocele, being the result of the deposition of cicatricial tissue over the protruding portion of Descemet's membrane. Ordinarily keratocele ruptures, incarceration or prolapse of the iris follows, with the subsequent formation of adherent leucoma.

Results of Keratectasia. Vision is disturbed not only on account of the opacification of the cornea, but also because of the irregularity of the curvature of the cornea affecting the pupillary area.

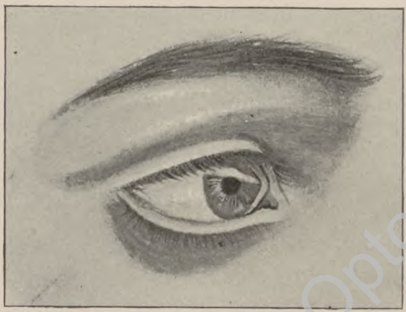
Treatment. Keratectasia may be treated as partial staphyloma, by incision followed by a compress bandage, ablation of a crescent-shaped piece of the corneal tissue, or by superficial cauterization. In cases where the opacity occupies a large portion of the pupillary area, iridectomy for reduction in tension as well as for visual purposes may be resorted to.

Keratoconus (Conical Cornea; Staphyloma Pellucida). (Fig. 149.) This condition resembles keratectasia; it, however, is unassociated with inflammation, and does not present opacification of the apex

until the condition has reached an advanced stage. It is due to thinning of the cornea and loss of power to withstand the intra-ocular tension. It begins between the ages of twelve to twenty years, and affects females more frequently than males. The condition develops in those who suffer from malnutrition, in those debilitated by illness, and occasionally in those who use the eyes excessively for close work.

Symptoms. Keratoconus is not accompanied by pain in its earliest stages. Its development is brought to the notice of the patient by the effect on vision. With the bulging of the cornea myopia and astigmatism are produced. The patient seeks relief, and is supplied with glasses which soon require change. With advance in the condition vision becomes much impaired, and because of the conical shape of the cornea relief cannot be obtained by glasses, as suitable glasses cannot be made to correct the peculiar curvatures produced. The change in the shape advances in many cases until the conical condition is

FIG. 149.



Conical cornea. (DALRYMPLE.)

very marked. The apex of the cone becomes opaque and the process limited. Spontaneous perforation and resolution do not occur in these cases.

Diagnosis. In the very early stage of keratoconus diagnosis is not easy, but careful examination by means of the ophthalmometer and by Placido's keratoscope makes the diagnosis possible.

Treatment. The use of glasses in the early stage is advisable. In the later stage improvement in vision may be obtained by the use of the stenopeic slit; Raehlman's hyperbolic lenses are of service in some cases. In advanced cases improvement in vision may be obtained by methods calculated to produce cicatricial contraction of the cornea. This may be brought about by puncture at the apex, by excision of a portion of the tissue at the apex, or by use of the cautery applied to the apex of the cone. Removal of a small circular portion at the apex of the cornea by the trephine has also been resorted to. Operative measures affecting the apex of the cornea increase the opacification, which is in almost every case

directly in the pupillary area. As a consequence it becomes necessary to make an eccentric pupil for visual purposes.

Buphthalmos (Hydrophthalmos; Keratoglobus; Megalo Cornea). This affection consists in a uniform enlargement of the globe. It appears at or shortly after birth, and is supposed to be due to congenital glaucoma. The condition usually affects both eyes. It progresses extremely slowly. In buphthalmos the diameter of the cornea is increased, the curvature of the cornea is less acute, the anterior chamber usually deeper. The lens frequently becomes dislocated, and as the disease advances vision is slowly reduced and sometimes entirely lost. In some cases the cornea remains clear (*keratoglobus pellucida*); in others it becomes cloudy, the condition then being known as *keratoglobus turbidus.*

Treatment is of little avail. The general condition of the patient should be improved as much as possible. Should the tension of the globe be increased, pilocarpine or eserine may be employed. At the best the prognosis is unfavorable.

Injuries of the Cornea. Injuries of the cornea of all degrees of severity occur. With abrasion of the cornea the patient suffers intense pain, at first of a smarting, burning character, and a feeling as of a foreign body in the eye. Photophobia is also experienced. The intense disturbance produced by abrasion of the cornea is due to the injury to the sensory nerve filaments that terminate in this layer. The symptoms persist from twenty-four to forty-eight hours; as the epithelium is regenerated they disappear. In incised wounds of the cornea the pain is relatively slight. Wounds of the cornea require treatment to prevent infection, consisting of careful cleansing with antiseptic solutions. Where the wound is an extensive one the margins may be approximated by means of suture.

Foreign Bodies in the Cornea. The prominent position of the cornea exposes it to the contact of foreign bodies of all kinds. When a foreign body strikes the cornea, if the sensitiveness of the cornea is normal, the eyelids close involuntarily, and if the foreign body is not fixed in the corneal tissue it will descend into the lower cul-de-sac or become fixed on the conjunctiva of the upper lid. If the foreign body remains attached to the cornea or imbedded in its tissues, a scratching sensation in the lids, usually the upper lid, is experienced, and this is repeated whenever the eye is opened or closed. The foreign body may be simply adherent to the epithelial layer, it may penetrate the epithelium and project from the surface, it may become totally imbedded in the cornea, or it may pierce the corneal tissue and project into the anterior chamber. Particles that enter the cornea do not, as a rule, produce discoloration of the corneal tissue; however, a hot cinder produces an eschar which, after the cinder is removed, appears as a circular ring of brown tissue.

The presence of a foreign body on the cornea is accompanied by photophobia, lacrymation, and often by pain that does not entirely depart even when the lids are kept quiet, and by pain referable to the

eyeball and to the corresponding side of the head. Soon injection of the ocular conjunctiva develops; this injection may be very mild, or it may be marked, varying according to the degree of irritation produced. If the foreign body is permitted to remain in the cornea, it may become loosened in a few hours or days and be removed from the cornea by the mechanical action of the lids. It may have carried micro-organisms into the tissues of the cornea, producing ulcer; the defect in the cornea may become invaded by germs from the conjunctiva, which, gaining entrance into the tissues of the cornea, may themselves set up a destructive inflammation.

Treatment. After instilling a drop (4 to 10 per cent. solution) of cocaine the removal of the foreign body should be attempted. Foreign bodies, if superficially imbedded, may often be removed by means of a small probang of absorbent cotton wound around the end of an applicator. If the foreign body is firmly imbedded, it should be lifted from the corneal tissue by means of a spud or sharp-pointed foreign-body needle. In certain cases it is necessary to cut the corneal tissue about the margin of the foreign body to get beneath it to lift it out. In cases where the foreign body has penetrated the cornea and projects into the anterior chamber, it is at times necessary to support it from behind while the tissues are cut away sufficiently to enable the surgeon to grasp it with a small forceps. After removal of the foreign body the defect in the cornea occasions the individual some annoyance until the epithelium has extended over the affected area. During this time the eye should be cleansed with an antiseptic solution from time to time (boric acid, 3 per cent.); and if infection is feared, more energetic antiseptic measures should be employed.

Blood-staining of the cornea sometimes follows injuries to the eye which result in hemorrhage into the anterior and posterior chambers. The pigmentation of the cornea is due primarily to the entrance of hæmoglobin in solution into the corneal tissue by way of Fontana's spaces, the hæmoglobin being the result of disintegration of red blood corpuscles. It permeates the lymph canalicular system of the cornea, and there undergoes a change into hæmosiderin, an insoluble product which is deposited in the corneal tissue in the shape of minute, irregular, octahedral crystals. These crystals occupy not only the spaces between the lamellæ, but also the spaces between the connective bundles and fibres of the cornea. The color of the cornea at an early stage is olive, with a tendency to red. This soon becomes a deep brown. The staining of the cornea occupies the central portion, reaching almost to the limbus in marked cases. At the limbus an annular ring of transparent cornea is found, measuring one-half to one and one-half millimetres in width. The transparent ring of the cornea is due to the removal of the hæmoglobin from the corneal tissue; also to the fact that the alkalinity of the cornea is maintained at the periphery through the influence of blood in the capillaries of the limbus conjunctivæ, preventing the change

into hæmosiderin. The staining of the cornea develops relatively slowly after the first appearance is noticed. It may take from one to four weeks for it to reach its height. In some cases the staining remains with little change for months, but in many absorption goes on slowly, and eventually the cornea may regain its transparency. Accompanying this condition we may have secondary glaucoma, loss of vision, and pain, depending not on the condition of the cornea, but on the condition of the eye itself.

Treatment. There is no treatment aside from stimulation by means of moist heat, that is of value in this condition. Frequently the condition of the eye is such that enucleation becomes necessary.

Burns of the Cornea. Burns of the cornea are not very infrequent. They are due to the entrance of molten metal, cinders, steam, acids, alkalies, burning gunpowder, etc.

Molten metal impinging upon the cornea seldom does much harm. The surface epithelium may be destroyed. The metal usually escapes from the eye at once or falls into the conjunctival sac, where the greater damage is done.

Glowing embers alighting on the cornea may become adherent and destroy the corneal tissue to some depth; ordinarily the burn is superficial.

Glowing cinders that fly into the eye sometimes become imbedded in the cornea, rendering necrotic the tissue that comes in direct contact with the cinder. Before recovery takes place, this necrotic layer of tissue must be cast off.

Steam entering the eye may destroy the epithelial layer in the portion of cornea exposed, usually a narrow strip lying in the horizontal meridian corresponding to the palpebral fissure. The treatment consists in cleansing the eye and instilling an antiseptic or aseptic oily preparation sufficiently often to protect the affected area, usually three to four times daily. Olive oil, with 5 per cent. of boric acid or vaseline, may be used.

Burns of the cornea from acid usually affect the entire surface of the cornea, turning it a grayish hue, destroying the epithelium, the superficial layers of which soon become detached. With burns of this character there are some secretion from the conjunctiva, increased lacrymation, and swelling of the lids, the appearance presented indicating a grave lesion. Except in rare cases, burns from acids are recovered from with little if any loss of tissue or permanent injury to the eye. The treatment consists in thoroughly cleansing the eye, using weak alkaline solutions (bicarbonate of sodium or very weak ammonia), and, as in burns due to thermal agencies, the instillation of an oily substance.

Burns of the cornea due to an alkali are most frequently occasioned by the entrance of quicklime into the eye. The fine particles of lime become imbedded in the corneal tissue, and the escharotic action is continued for some time. When first seen, the affected area presents a grayish discoloration, frequently not

very dense. The appearance of the cornea after combustion from *lime,* as first presented, usually induces a more favorable prognosis than is warrantable. The opacification in almost all cases increases in intensity as the healing process advances. Treatment in these cases is to free the cornea as rapidly as possible from particles of lime by washing with olive oil, which is probably the most suitable for this purpose. After the particles of lime have been removed, the eye should be filled with syrup of cane-sugar, as sugar forms an insoluble compound with lime, preventing extension of the destructive process. The subsequent treatment of burns from lime is like that of burns with acids and thermal agencies.

Tumors of the Cornea. Tumors of the cornea originating in the cornea are extremely rare. Cysts sometimes develop, but they are the result of inflammatory processes, are usually extremely small, and seldom require treatment. Tumors of the cornea usually extend onto the conjunctiva; they are dermoid, papilloma, fibroma, epithelioma, and sarcoma. These have all been treated of in the chapter on diseases of the conjunctiva, and need no further mention.

SCLERA.

The sclera, together with the cornea, forms the fibrous coat of the eye. It is the segment of a sphere, the radius of curvature of which is about 12 mm. At its junction with the cornea a depression is found, which is known as the sulcus scleræ. The sclera is thickest at its posterior part, where it measures about 1 mm. in thickness; thinnest near the equator, becoming slightly increased in thickness in its anterior portion, where it receives the insertion of the recti muscles. The anterior portion of the sclera is pierced by a number of minute openings through which pass the anterior ciliary arteries and veins. Some small nerve twigs also pass through the sclera a short distance back from the corneal margin. At the equator of the globe the sclera is pierced by four, sometimes five, relatively large openings, which give passage to the large venæ vorticosæ. Posteriorly a number of openings are found which give passage to the short ciliary arteries and ciliary nerves, and a large opening to permit the passage of the optic nerve fibres. This opening, which measures about 1.5 mm. in diameter, is traversed by connective-tissue bundles, which are continuous with the sclera proper, forming what is known as the cribriform plate. The sclera is made up of connective-tissue fibres, which are not disposed in regular lamellæ as are those of the cornea, and which run in various directions. Between these bundles of connective-tissue fibres spaces are found resembling the lacunæ of the cornea. In the sclera about the optic nerve entrance and in its anterior portion branching pigment cells are found. These are most plentiful near the inner surface of the sclera. In certain individuals the openings for the passage of the anterior ciliary veins

are pigmented, giving the appearance of a number of black points on the sclera. In certain individuals, particularly those of the colored race, the pigmentation of the sclera, anteriorly, is very marked.

At its anterior margin the tissue of the sclera is continuous with that of the cornea. Near the anterior margin of the cornea, and separated from its inner surface by a thin layer of connective-tissue bundles, is the venous sinus known as Schlemm's canal. Externally the sclera is covered by the visceral layer of Tenon's capsule. Internally it affords at its anterior part attachment for the ciliary body by means of the ligamentum annularis. Posteriorly the inner surface of the sclera is covered by the lamina fusca of the choroid, the sclera being separated from the choroid proper by the suprachoroidal lymph space. But few bloodvessels are found in the substance of the sclera. The episcleral tissue, however, is richly supplied with bloodvessels, particularly in its anterior portion. The nerve supply of the sclera is extremely scanty. Posteriorly the tissue of the sclera is continuous with the sheath of the optic nerve.

Scleritis. Inflammations of the sclera are of relatively rare occurrence, and may be divided clinically into two forms: episcleritis, (*a*) fugacious and (*b*) persistent, and deep scleritis.

Fugacious Episcleritis. A transient inflammation of the episcleral tissue sometimes occurs, the attack being characterized by the appearance of an injected area with slight elevation of the conjunctiva, the disease usually occupying from 1/6 to 1/5 of the surface of the sclera in its anterior segment. The appearance of the inflamed area is accompanied by symptoms of irritation, manifested by an increase of lacrymation, perhaps very slight mucoid secretion, slight pain in the eyeball radiating to the temple and forehead, and photophobia. The irritation is increased by use of the eyes for near work. This inflammation of the episcleral tissue reaches its height in from three to four days, and then gradually subsides, every trace disappearing at the end of a week or ten days.

The affection is met with in individuals at the age of puberty and in early adult life, and is prone to recur when the system is in a run-down condition.

Cause. Episcleritis of this nature is met with most frequently in those who present a rheumatic or uric-acid diathesis. Undue use of the eyes, eyestrain consequent on imperfectly corrected errors of refraction and imbalance of the ocular muscles, exposure to inclement weather, use of the eyes in a bright light, disturbance of digestion, all may contribute to bring on an attack.

Treatment. Treatment consists in correcting any error of refraction that may exist, in correcting the condition of the system which predisposes to attacks, and in protecting the eyes from the influence of bright light when this is a factor in the production of the disturbance.

Episcleritis is characterized by inflamed nodules which occur near the margin of the cornea. The area of inflammation is often single.

The tissue at the points affected is slightly raised, and is of a deep-red or purplish hue. The bloodvessels of the conjunctiva overlying the inflamed area are enlarged and injected. The inflammation advances slowly, and is attended by symptoms of irritation that are much more severe in some cases than in others, and rather deep neuralgic pain with photophobia is also experienced. Both eyes usually are attacked, and fresh areas may be involved before subsidence of the process in the part first affected.

Duration. The affection persists from four to eight weeks, but may continue for a longer period. Recurrences are the rule, and another attack may occur immediately after the subsidence of one attack, or may not occur until years have elapsed.

As a result of the inflammatory process, there is usually a slight bluish-colored patch in the sclera, but recovery may take place without leaving a trace. This disease usually attacks adults, but may occur at the period of adolescence.

Cause. Rheumatism and gout are common causes. In some cases the cause is obscure. It is probable, however, that digestive disturbances are accountable for the greater number of cases.

Prognosis. The prognosis is favorable, as a rule. In rare cases ectasia and inflammation of the deeper structures may result.

Treatment. Reuss advocates the use of the constant current. Scarification has been advised in cases where pain is severe, and ointments of various kinds are indicated. The process is most favorably influenced by internal medication, consisting of the salicylates, iodide of potassium, and the mercurials in small continued doses.

The deep form of scleritis cannot readily be distinguished in its onset from the superficial form, except in degree of severity. Pain accompanying deep scleritis is usually more severe. The affected area is of a deep-purple hue. The elevation is somewhat more pronounced. The affected area is larger, and may extend around the entire cornea.

Accompanying deep scleritis we frequently have inflammation of the cornea in the vicinity of the affected area, and the deeper structure of the eye—iris, ciliary body, and anterior portions of the choroid—also are affected. The process attacks both eyes and progresses extremely slowly.

The change that takes place leads to attenuation of the tissue of the sclera, reduces it in thickness, and lessens its power of resistance, so that it cannot withstand the normal intra-ocular pressure, and becomes ectatic. The bulging of the sclera is usually irregular; it seldom extends around the entire cornea, but in the few cases in which this does occur the entire cornea is pushed forward. The ectasia usually occurs after subsidence of the inflammation. When the scleritis subsides the affected area presents a dark-blue appearance on account of thinning, which permits the pigment of the uveal tract to show through. Accompanying the formation of

these staphylomatous protrusions disorganization of the interior of the globe is observed. Vision is ordinarily very much impaired.

Sclero-kerato-iritis (Scrofulous Scleritis; Anterior Uveitis). This condition is one closely allied to deep scleritis, but differs from it in that the whole anterior segment of the globe is affected. The inflammatory process usually begins at or near the sclerocorneal junction, and progresses as a deep scleritis involving the cornea (which takes on a condition of sclerosis), the iris, the ciliary body, and the anterior portion of the choroid. In addition to the appearances and symptoms that accompany scleritis and sclerosing keratitis, symptoms peculiar to involvement of the anterior portion of the uveal tract are present. The iris becomes congested, loses its transparency, takes on a dusky hue, and is thickened. The aqueous humor becomes turbid as a result of transudation of plastic lymph from the bloodvessels of the iris and ciliary body. There is pain referable generally to the temple and forehead. The anterior portion of the vitreous body becomes filled with flocculi consisting of fibrin. The disease progresses very slowly, both eyes being commonly affected. Individuals in early childhood and at the age of puberty are most frequently attacked. They are individuals with inherited syphilis and those whose condition may be described by the term scrofulous. As a result of sclero-kerato-iritis, sclerosis of the anterior portion of the sclera and of the cornea occurs, followed in many cases by sclero-ectasiæ. The ordinary results of severe iritis are also present. The choroiditis is followed by atrophic changes in that membrane. The ciliary body becomes atrophic and much elongated by the stretching that accompanies the ectatic process. The cornea is thinned throughout the area involved in the sclerosis and becomes more or ess opaque.

The effect on vision is pronounced, the diminution depending on the degree of opacity of the cornea and the interference with the transparency of the media of the eye. As a result of this process the crystalline lens not infrequently becomes opaque, shrunken, and the site of calcareous deposits. In consequence of the changes affecting the filtration angle, increase in the tension of the eyeball develops, secondary glaucoma results, and total loss of vision may follow. The increase in tension may also lead to spontaneous rupture of the glope, the rupture occurring at some point in the ectatic portion.

Treatment. Treatment directed to the correction of any dyscrasia of the system that may exist should be instituted. If the condition accompanies hereditary syphilis, antisyphilitic remedies should be employed. If a uric-acid diathesis is present, it should be corrected. Local treatment consists in the endeavor to prevent the formation of posterior synechiæ. Hot bathing with a solution of boric acid and the introduction of a mercurial locally are of service. Ointments of the yellow oxide and mercuric chloride appear to be best suited. As a matter of fact, local treatment seems to

have little effect in arresting the progress of this condition. In eyes affected by this disease more or less serious damage is wrought.

Scleral ectasiæ (staphyloma of the sclera) are classified as anterior, equatorial, and posterior. Anterior ectasiæ may be single or multiple. They may be annular, extending entirely around the periphery of the cornea. At the equator the ectasiæ may present the same conditions present in the anterior segment. Posterior ectasiæ are usually single, and frequently include the optic nerve entrance. Scleral ectasiæ present a bluish appearance, because of the pigmented uvea, which shows through the thinned sclera.

Cause. Staphylomata are produced either because of a reduction in the power of the sclera to withstand the normal intra-ocular pressure or tension, weakness of the scleral coat being either inherent or the result of disease; or it is due to an increase in the intra-ocular tension above the normal and above the power of the sclera to withstand.

Scleral ectasiæ are most frequently due to scleritis. When intra-ocular pressure only is the cause, equatorial staphyloma usually results. If the weakness is structural and congenital, the staphyloma usually occurs at the posterior pole. Staphyloma not infrequently accompanies neoplasms of the interior of the eye. In certain cases ectasia of the sclera reaches enormous dimensions, as in the case of scleral cyst accompanying microphthalmos.

Treatment. After ectasiæ of the sclera have developed, treatment is of no avail. As a prophylactic measure in inflammatory conditions which have resulted in the formation of posterior synechiæ, iridectomy may be useful, and in cases of glaucoma, either primary or secondary, the same procedure may prevent development of ectasiæ by reducing the intra-ocular tension.

Syphilis of the Sclera. Syphilitic involvement of the sclera is seldom observed. When it does occur, it manifests itself in the form of gumma, usually affecting the anterior segment of the sclera. Cases have been observed in which the posterior portion of the sclera has been the seat of a gummatous mass. When gumma of the sclera occurs in a visible portion, it presents itself first as a small nodule simulating a large phlyctenule. It increases in size quite rapidly, the elevation becoming pronounced, the base of the elevation being deep red in color, and the congestion extending for some distance into the surrounding tissue. The apex of the elevation is of a yellowish hue. The growth is circular at its base. It may reach a diameter of 1 cm. to 2 cm. If treatment is not instituted, ulceration takes place at the apex, due to breaking down of the tissue, and destruction of the eye may ensue. The tumor is rather firm in consistence, and is elastic. It may be mistaken for sarcoma. A microscopical examination of excised parts may disclose the presence of cells which closely resemble those of sarcomatous tissue. Sarcoma of the sclera as a primary disease is extremely rare, and the presence of a growth such as has just been described should always awaken suspicion of

a syphilitic origin. The history of the case is not always to be relied upon to substantiate the diagnosis. Gumma of the sclera is seldom met with in children, but is an occasional manifestation of tertiary syphilis in adults. The writer has never seen a process of this kind occurring as a result of inherited syphilis. In these cases the tissue of the sclera is invaded by a small-cell infiltration. Fibres of the sclera are pressed apart, and some disappear absolutely. As the process subsides, if scleral tissue has been destroyed, it is replaced by cicatricial tissue.

Treatment. Under vigorous antisyphilitic treatment of the usual kind gumma of the sclera subsides with marvellous rapidity, and, if the deeper tissues of the globe are not involved, no trace of the tumor is left.

Tumors of the sclera other than that just mentioned do not occur as primary growths.

Injuries to the Sclera. The sclera is subject to injuries of various kinds: perforating wounds, incised wounds, lacerating wounds, and rupture of the sclera. Perforating wounds of the sclera, if made with sharp and non-infected instruments, are usually of little importance, provided the perforation be small and occur back of the ciliary region. If, however, the wound is large, permitting prolapse of a portion of the ciliary body, the effect on vision may be disastrous. Non-infected wounds heal rapidly. It occurs from time to time that perforating wounds of the sclera result in total detachment of the retina without suppuration and without inflammatory reaction of any appreciable degree. A case in point is that of a woman who, when shaking a carpet, felt a twinge in the eye, and found that a carpet tack had pierced the sclera about 7 mm. from the sclero-corneal margin. She removed the tack by traction and presented herself at the hospital within twenty-four hours. At that time a small opening could be detected in the sclera, into which a bead of vitreous projected. There was but slight injection of the sclera and conjunctiva, and the opening in the sclera had already become closed by plastic lymph. Examination with the ophthalmoscope disclosed the point of entrance of the tack. There was no hemorrhage in the vitreous and very little blood surrounded the opening. The eye was bandaged and the patient removed to her home. No inflammation resulted. In the course of four weeks complete detachment of the retina developed.

Perforating wounds of the sclera may result in infection of the eyeball and loss of the globe by panophthalmitis.

Lacerating Wounds. Lacerating incised wounds of the sclera, if they occur posterior to the ciliary region and are not of great extent, may be closed either by a scleral or conjunctival suture, and recovery may occur with little or no loss of vision. Clean incised wounds of the sclera, even those which involve the ciliary region, may also heal, if properly closed, without loss of vision. In the latter form of wound in the sclera the prolapse of vitreous and prolapse of the

ciliary body, if they occur, should be excised. The wound may then be closed by scleral or conjunctival suture.

Lacerating wounds affecting the ciliary body result in almost every case in loss of the eye. Removal of the eye, frequently, is not at once necessary; but as the scar contracts painful symptoms develop, and, in some cases, symptoms of sympathetic disturbance of the other eye become pronounced and render enucleation necessary.

All cases of wound to the sclera should be treated on strictly antiseptic principles. Prolapsing tissue should be excised if an attempt is to be made to save the globe. Under ordinary circumstances the patient, whether suffering from a perforating wound, an incised or a lacerating wound of the globe, should be put to bed, and compress bandages applied until recovery is well advanced.

Rupture of the Sclera. Rupture of the sclera occurs in the majority of cases within the zone included in a strip 1 cm. wide from the margin of the cornea. A blow on the eye from whatever source may produce such a rupture. The rupture takes place above most frequently. It occurs in the shape of an irregular line extending from near the margin of the cornea diagonally backward, and measures in the majority of cases about 2 cm. in length. The ciliary body and the anterior portion of the choroid are usually pushed into the wound, and present as a dark line following the wound; while the crystalline lens may be forced out of the eye and lie beneath the conjunctiva. Subconjunctival hemorrhage occurs, which extends to some distance from the wound, sometimes extending beneath the entire ocular conjunctiva. The anterior and vitreous chambers are more or less filled with blood. Such wounds frequently occur without rupture of the conjunctiva.

Rupture of the sclera is not necessarily confined to the anterior segment of the globe. In rare cases it occurs in the posterior segment of the globe. It is then manifest by a greater or less degree of exophthalmos due to the presence of blood in the orbital tissues. Cases in which the rupture is very slight may result in recovery without loss of vision and without a painful globe, but such are very rare.

In the early stage of rupture of the sclera in the ciliary region pain may be relatively slight, and, to the inexperienced, the prospects of a favorable recovery may appear bright. However, eventually the conditions above described almost invariably develop, and enucleation must be resorted to.

Foreign Bodies in the Sclera. Foreign bodies in the sclera are seldom present without perforation and entrance into the posterior chamber of the eye, the result to the sclera being usually a wound simply. When the body has remained imbedded in the sclera, it should be removed, its removal being effected by the means ordinarily employed to remove foreign bodies from other tissues of the body.

CHAPTER VII.

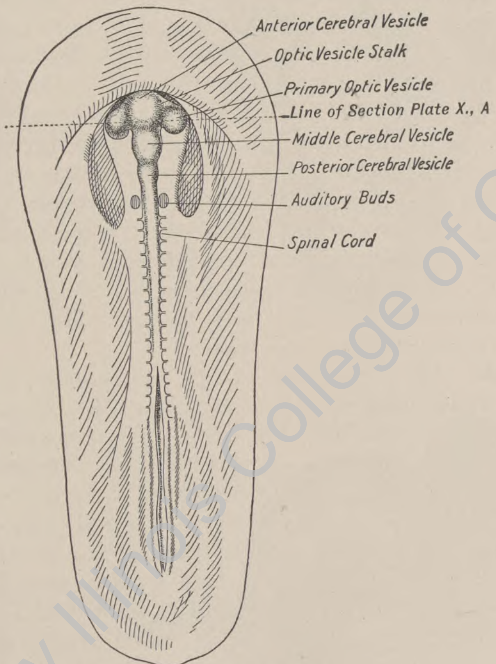
EMBRYOLOGY OF THE EYE; ANOMALIES, DISEASES, AND INJURIES OF THE IRIS, CILIARY BODY, CHOROID, AND VITREOUS.

BY H. V. WÜRDEMANN, M.D.

EMBRYOLOGY OF THE EYE.

General Development. The eye begins its development as a budding of the mesodermic and ectodermic layers of the embryo, called the primary optic vesicle, connected with the brain by the optic pedicle

FIG. 150.



Entire embryo of a frog. (After FICK.)

or stalk, which subsequently becomes the optic nerve. (Fig. 150.) The development of the mesodermic portion is so slow in the majority of embryos that the vesicle is not fully covered by this structure,

and thus remains in intimate connection with the ectoderm, being at first, histologically, very much like the brain vesicle. (Plate X., A.)

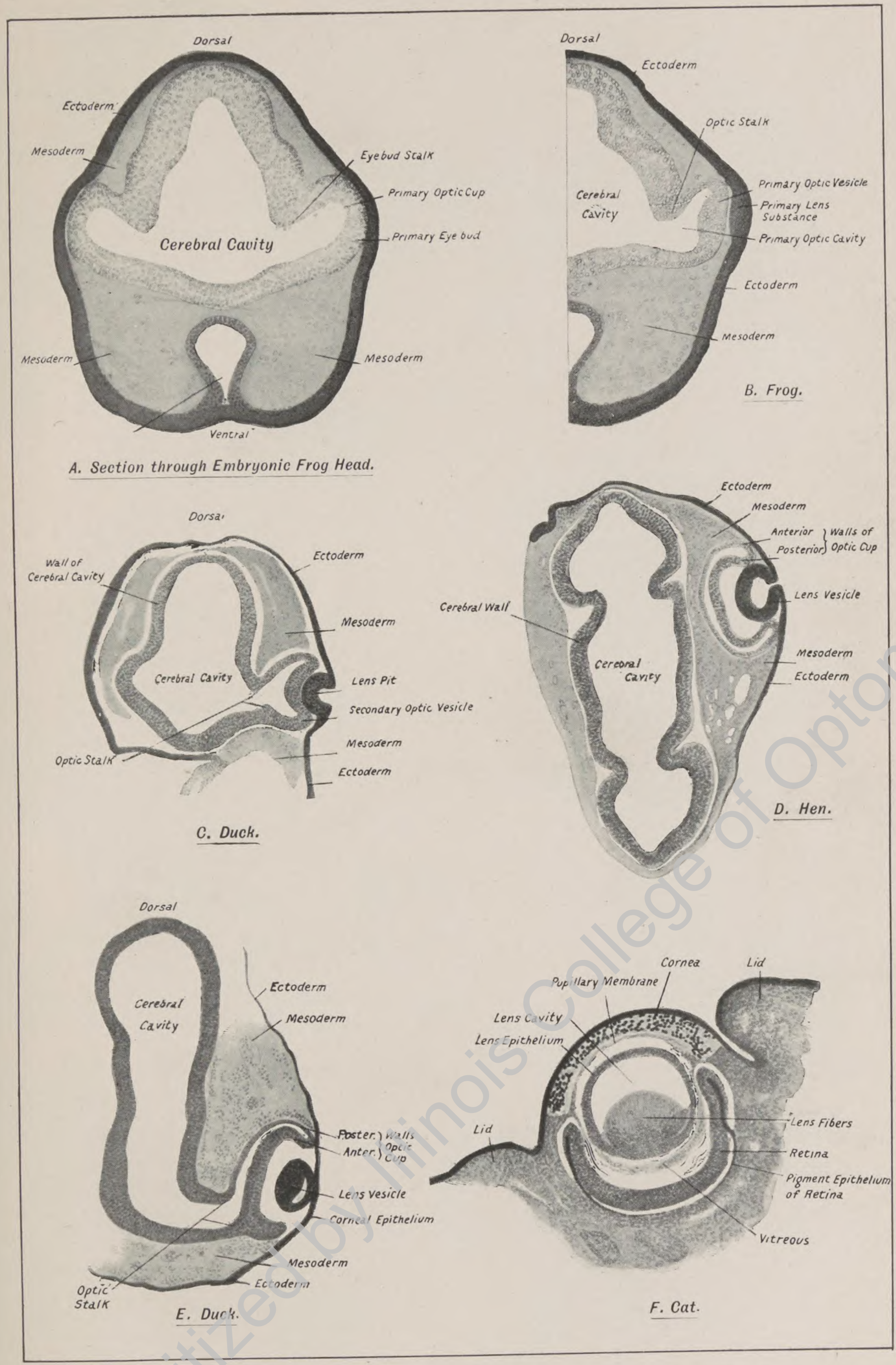
The primary optic vesicle then becomes thicker, becoming invaginated, forming the primary optic cup (Plate X., B); the ectoderm lying over the eyebud becomes thickened, and at the same time sinks into the subjacent invaginated optic vesicle, forming a depression known as the lens pit or secondary optic cup. (Plate X., C.) The mesoderm does not cover this place, but the ectoderm thickens, ultimately forming the lens. At this time the eye has somewhat the form of a pair of scissors, the points projecting upward. (Plate X., D.) These points soon coalesce, covering the secondary cup, closing over the lens, and developing the cornea. (Plate X., E.)

As development goes on, the invaginations progress with uniform rapidity until the lens sac reaches completion, when the expansion of the inner wall of the latter no longer keeps pace with the pushing in of the optic vesicle, and thus forms a space, the primitive vitreous chamber. (Plate X., F.) The portion of the wall invaginated by the process of involution undergoes proliferation, forming the retinal layer. The outer layer of the optic vesicle increases, but becomes so attenuated that by the time the retinal layer comes into contact with it, it is so thin that it forms ultimately but a single layer of cells, the retinal epithelium. (Plate XI., A, F, G.) The inner wall thickens and forms the essential nerve elements of the retina. The lower portion of this double-layered vesicle is not completed, but forms a hiatus, the fœtal ocular or choroidal cleft, which soon narrows and finally closes, but, before doing this, allows the entrance of mesodermic tissue, which constitutes the primary vitreous stroma. The imprisoned mesodermic tissue in the optic stalk is represented later by the central vessels of the retina and the associated connective tissue of the optic nerve. The optic stalk becomes the optic nerve, chiasm, and tract,

Lens. All of the lens, except the capsule, is of ectodermic origin. Early in development the optic cup closes (Plate X., E), the inner wall becoming thicker than the outer. The thickening progresses, so that by the time the lens sac has become completely isolated from its attachment to the surface ectoderm its walls consist of two or three layers of epithelial cells, limited externally by a delicate membrane, the lens capsule. The obliteration of the cavity of the lens sac and the conversion of the organ into a solid mass are effected by a phenomenal growth and elongation of the epithelial elements composing its posterior or internal wall, which rapidly increases in length, becoming converted into the primitive lens-fibres. (Plate XI., A.)

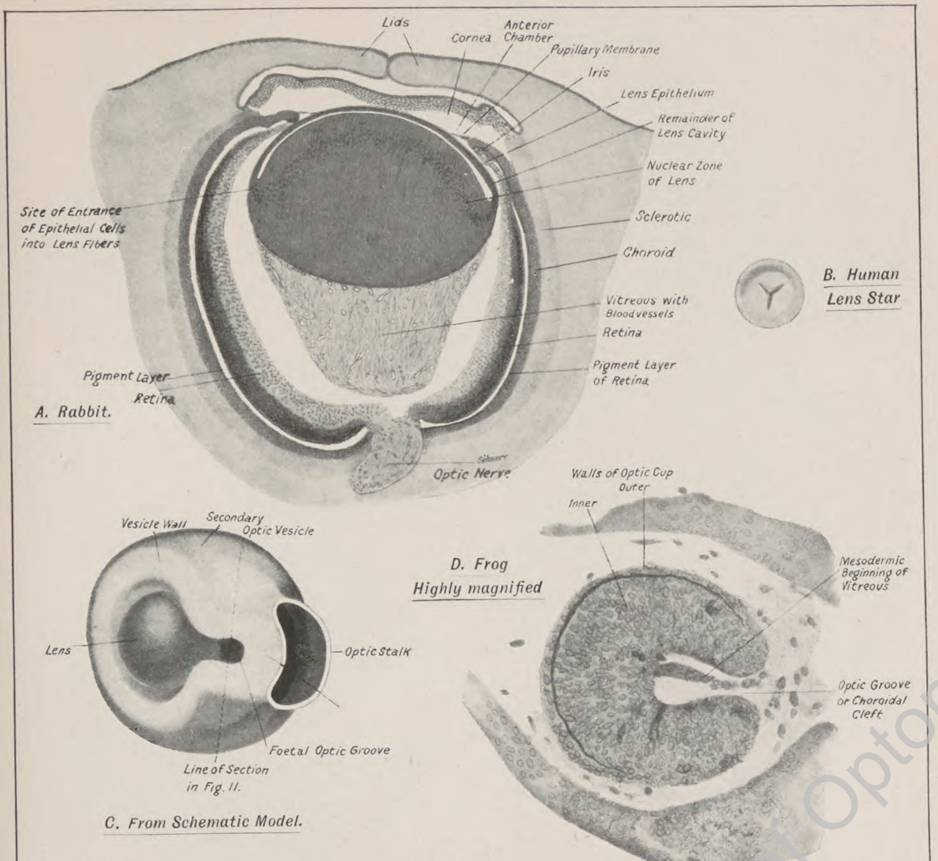
From the unusual demands made by the young, rapidly growing, and non-vascular lens on the surrounding tissue for nutrition, a special temporary structure develops, the tunica vasculosa lentis (Fig. 152), which completely surrounds the young lens from the second month toward the end of gestation, at which period it usually becomes

PLATE X.



Development of the Eye. (Fick.)

PLATE XI.



Development of the Eye. (Fick.)

atrophied and disappears; when persistent, it is called persisting pupillary membrane. (Plate XIII., Fig. 12.)

Vitreous. The vitreous body is composed of connective-tissue elements formed from the mesoderm. In the process of development a bud of mesoderm is pushed into the eye through the choroidal cleft, which soon grows and acquires bloodvessels. Through these vessels leucocytes and round and spindle cells are produced; the former have amœboid movements, and the latter are fixed cells. These actively proliferate, filling the space between the primitive lens and the retinal layer of the optic cup, forming the substance of the vitreous.

Bloodvessels. The bloodvessels of the eye are developed from ingrowths of mesodermic tissue. Coincidental with the growth of the primitive vitreous an artery and vein develop in the optic nerve,

FIG. 151.

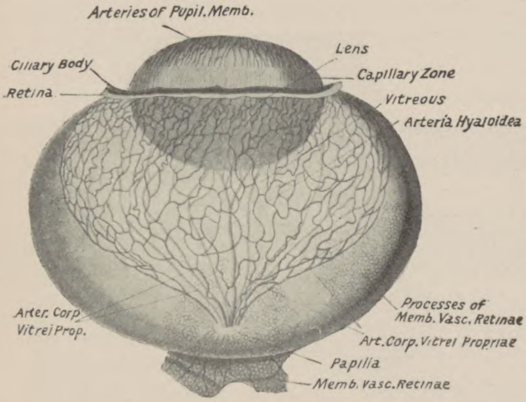


FIG. 152.

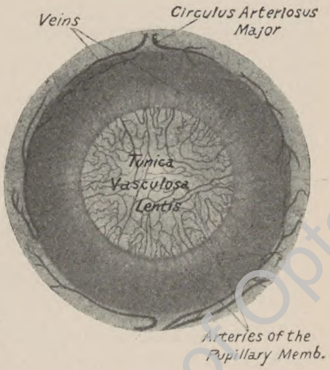


FIG. 151.—Bloodvessels of the embryonic eye. Pig embryo. Magnified 7½ diameters. (After SCHULTZE.)

FIG. 152.—The pupillary membrane and bloodvessels of the iris. Pig embryo. The arteries springing from the circulus arteriosus of the pupillary membrane are first visible in the pupil. (After SCHULTZE.)

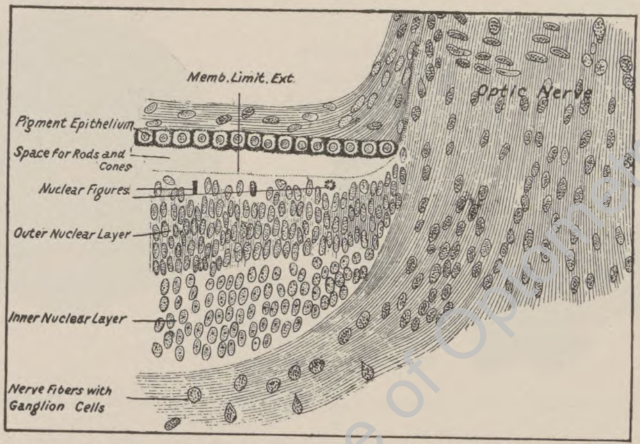
which later become the arteria centralis retinæ and veins; an artery develops in the vitreous (the hyaloid), going to the posterior pole of the lens. Then the vitreous becomes abundantly supplied with capillaries (Plate XI., A), which new blood supply permits of increased nutrition, resulting in rapid proliferation of the mesodermic vitreous and growth of the lens. During the last weeks of fœtal life the bloodvessels of the vitreous and the lens disappear, leaving a passage, the hyaloid canal. If the hyaloid artery does not fully atrophy, vestiges may be seen later in life by the ophthalmoscope.

Retina. The retina is formed from the eye cup by early differentiation of the outer and inner layers. By the time the infolded portion of the vesicle has become closely attached to the outer segment it has increased many times in thickness (Plate XI., D, F, G); the

latter thins, but early accumulates pigment cells, first at the anterior pole, which gradually extend to the posterior pole, ultimately becoming the pigmented retinal epithelium. (Plate XI., A.) The proliferation of the inner layer results in the formation of two varieties of tissue, the nervous and the sustentacular tissue. The nervous layer differentiates into the nerve cells, their outgrowths, and the epithelium. The latter eventually forms the outer nuclear layer and the rods and cones, these two constituting the vision cells. (Fig. 153.)

The sustentacular tissue extends through the thickness of the retina and gives support to the nervous elements forming the radial fibres of Müller. Besides this, there are outgrowths of true connective tissue coming from the surrounding mesoderm, which accompany the ramifications of the retinal arteries; all this portion forms the pars optica retinæ. The anterior marginal zone of the optic cup becomes a thin, deeply pigmented layer of epithelium, covering the

FIG. 153.



Development of the retina. Human embryo. 3.8 mm. long. (After FALCHI.)

ciliary body, pars ciliaris retinæ, and the posterior surface of the iris, pars iridica retinæ. The anterior edges of the cup form into ridges, becoming the ora serrata.

Optic Nerve and Tracts. The anterior portion of the optic stalk becomes the optic nerve, the middle portions of either side unite to form the chiasm, and the posterior portions become the optic tracts. At first the optic stalk is a short tube between the primary optic vesicle and the mid-brain. (Plate X., A, B, C, D.) The invagination affecting the lower wall of the optic vesicle forming the choroidal fissure affects the optic stalk at the ocular end, allowing the entrance of vascular mesoderm, from which arise the retinal bloodvessels. As the lower wall of the stalk folds in, its lumen becomes obliterated by apposition of its walls and thickening due to active proliferation, the young optic nerve becoming solid, the imprisoned mesoderm

producing the accompanying bloodvessels and the connective tissue surrounding them. The nerve fibres are developed from neuroblasts passing from the retina toward the brain and others growing from the brain toward the retina. The sheaths of the optic nerve and the septa are produced by continuation of the mesoderm, which forms the cerebral dura, arachnoid, and pia.

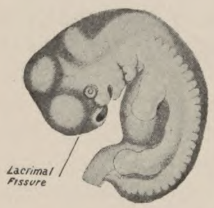
Uveal Tract (Choroid, Iris, and Ciliary Body). The large amount of vascular tissue in the iris, ciliary body, and choroid shows that the tunica vasculosa oculi or uveal tract has been developed from the mesoderm. In the early differentiation of the eye structures the lens sac becomes separated from the overlying ectoderm by a thin stroma of mesodermic tissue, which becomes cleft in development (Plate XI., D), one part remaining over the outer surface of the lens, and the other adhering to the inner surface of the ectoderm. The former constitutes the pupillary membrane and the latter the substantia propria of the cornea, the space between forming the anterior chamber. The forward growth of the thin double-layered lip of the optic cup beyond the edge of the lens and over its anterior surface forms the ciliary body and the iris; extending backward, it forms the primitive choroidal stroma which accompanies the retinal tissue in its growth forward. Almost the whole of the anterior surface of the lens becomes covered, with the exception of a central area corresponding to the pupil, which is closed first by the vascular pupillary membrane. (Figs. 151 and 152.) Further attenuation of the epithelial cells on the edge of the lips of the optic cup forms the columnar and cuboidal elements of the pars ciliaris and pars iridica retinæ. The pigmentation of the cells increases until the anterior portion of both layers forms the conspicuous pigment of the posterior surface of the iris and the ciliary body. About the third month of fœtal life the epithelial cells surrounding the equator of the lens form into a series of radial folds, into which delicate processes of mesodermic tissue extend, developing into the vascular structures of the ciliary processes. The outer stroma of the pars ciliaris becomes pigmented, the inner layer remaining uncolored.

Cornea and Sclera. With the exception of the corneal epithelium, the lens, and the nervous tunic with its cerebral attachments, all of which are derived from the ectoderm, the other parts of the eyeball are developed from the mesoderm surrounding the primary optic vesicle. At the same time that the many changes hitherto described occur in the optic vesicle the surrounding mesoderm exhibits marked proliferation and condensation, resulting in the production of a distinct envelope of embryonic connective tissue. The posterior segment of this mesodermic capsule differentiates late in fœtal life into an outer dense tunic, which becomes the sclerotic coat; the anterior portion becomes earlier differentiated into the substantia propria of the cornea, being developed from a homogeneous mass which fills the small cleft between the anterior pole of the lens and the ectoderm

which forms the corneal epithelium. Between the anterior surface of the lens and this mass, mesodermic cells go in and form the endothelium of the cornea. (Plate XI., F, G.) These cells throw out projections piercing the mass and forming the corneal corpuscles; the posterior layer of the homogeneous mass remains, forming the lamina elastica posterior, and the anterior portion forms the lamina elastica anterior. Precorneal bloodvessels develop later, but disappear before birth. (Plate XI., A.)

Ocular Appendages (Eyelids, Conjunctiva, Muscles, Glands, and Orbital Tissues). The eyelids develop early as an upper and a lower fold of the ectoderm, which grow over the corneal surface until they meet and fuse, this taking place early in the third month of fœtal life in man, continuing until shortly before birth, when the permanent separation is effected by cleavage along the line of juncture. (Plate XI., A.)

FIG. 154.



Human embryo of thirty-one days. Magnified 5 diameters. (After HIS.)

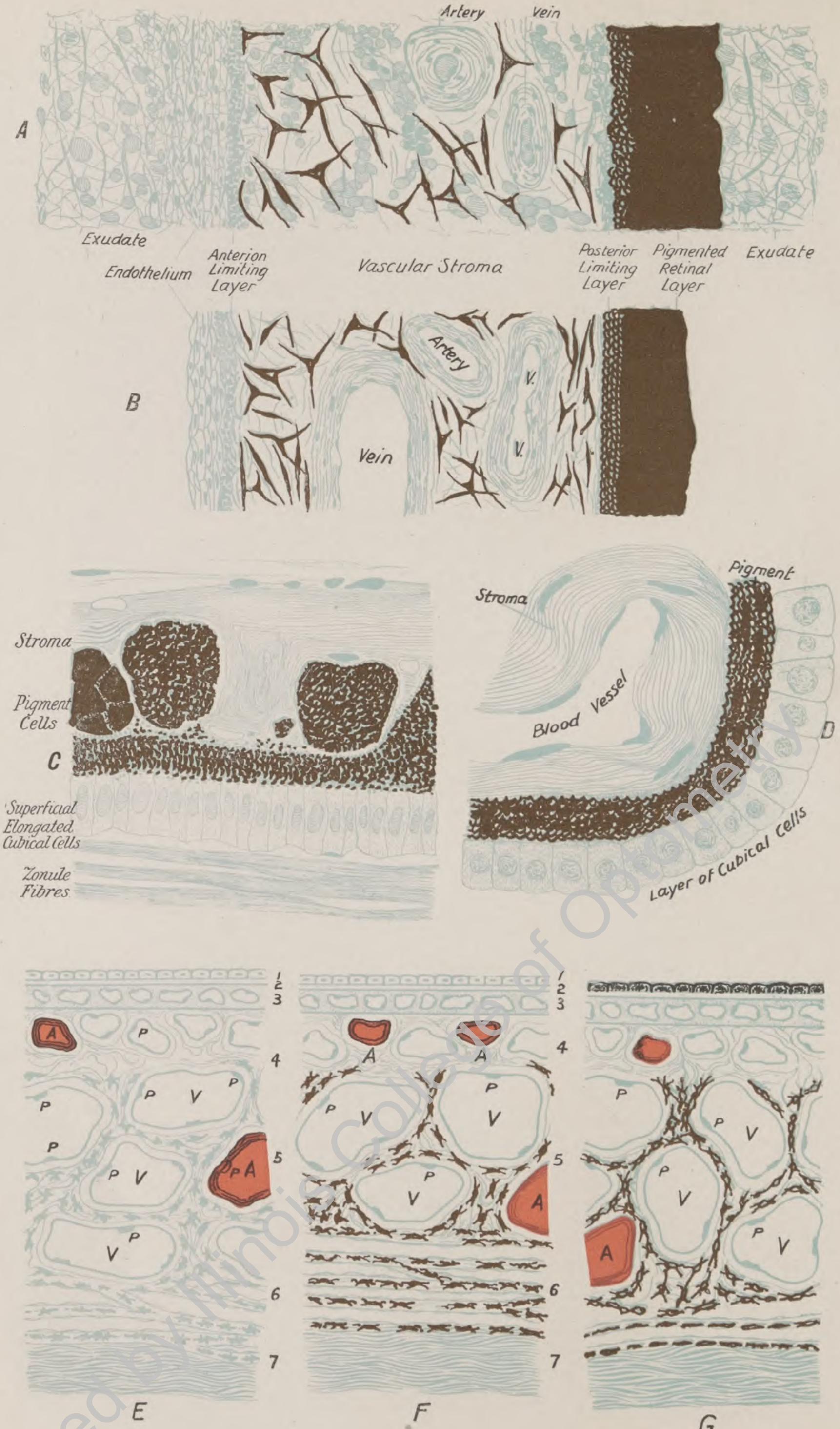
The hairs, the glands, lymphatics, tarsal and bulbar conjunctivæ, and the anterior epithelium of the cornea, are developed from the ectoderm. The lacrymal passages appear early as a fissure (about the thirtieth day), being developed, as are the tear glands and tear sacs, by infoldings of the ectoderm. (Fig. 154.) The ocular muscles, together with Tenon's capsule, the connective tissue, and various structures within the orbit, with the exception of the nerves, are derived from the mesoderm.

ANATOMY AND PHYSIOLOGY OF THE UVEA.

If the outer coat of the eyeball, which is composed of the cornea and sclera, be removed, a grape-like body is exposed, which is the uvea or middle coat of the eyeball. The anterior portion is composed of the iris, which is a diaphragm in front of the lens with a central opening forming the pupil; it extends to the junction of the cornea and sclera, where it is continued as the ciliary body; this being seen on cross-section is triangular in shape, and is a circular organ about 2 mm. wide, which is continued posteriorly as the choroid to the opening in the sclera which admits the optic nerve. The whole uvea is soft and friable, the choroidal portion being composed mainly of connective tissue and bloodvessels, whose function is to cover and nourish the essential parts of the eye. The anterior portion or iris is a photostat, and has, in addition, muscular elements; the ciliary body is composed of nervous, vascular, and muscular elements which have to do with secretion, excretion, and the function of accommodation.

Iris. Macroscopic Anatomy. The iris is a membranous and muscular diaphragm containing a central opening, the pupil. It extends

PLATE XII.



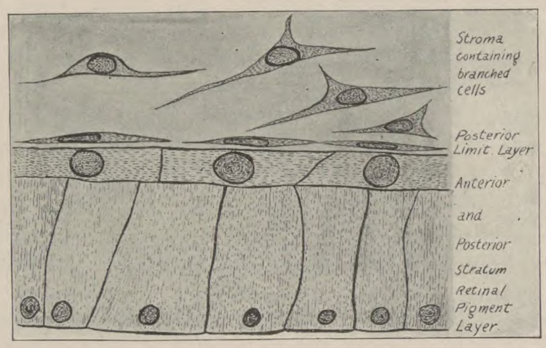
Diagrammatic Sections of Choroid.

A. Microscopic Section of Iris in Iritis. B. Microscopic Section of Normal Iris. C. Superficial Layers of the Flat Portion of a Ciliary Process in Meridianal Section. D. Meridianal Section of Portion of Ciliary Process near Apex. E. Albinotic Eye; no Pigment in Pigment Cells. F. Tessellated Fundus; Pigment Confined to Stroma. G. Negroid Fundus; Deeply Pigmented in both Retinal Layer and Stroma. A. Arteries. V. Veins. P. Perivascular Lymph Spaces.

from the anterior surface of the ciliary body over the lens; its central or pupillary border lies and glides upon the anterior capsule of the lens, thus obtaining a firm support. The ciliary border or root of the iris is more posterior, on account of the shape of the lens, and thus the iris forms a shallow truncated cone; its ciliary border is separated from the lens by a space, the posterior chamber. If the lens be absent, the iris loses its support, is tremulous (iridonesis), and extends in a plane. For normal appearance of the iris, see Fig. 6.

Microscopic Anatomy. On section the iris is seen to be composed of several distinct layers (Plate XII., A): 1. Anterior endothelium. 2. Anterior boundary layer. 3. Vascular stroma layer. 4. Posterior limiting layer. 5. Pigment layer, composed of (*a*) the outer or anterior layer of pigmented spindle cells, and (*b*) inner or posterior layer of pigmented polygonal cells.

FIG. 155.



Posterior layers of the iris of an albinotic human eye. Magnified 350 times. (After FUCHS.)

The stroma of the iris consists of numerous bloodvessels enclosed in a thick adventitia, which run in a radial direction from the ciliary to the pupillary margins, and are surrounded by a loose meshwork of branched and pigmented cells. There is a flat band of smooth muscular fibres lying close to the posterior surface of the iris and near the pupillary margin, which composes the sphincter muscle or constrictor pupillæ. On the anterior surface is a dense layer of cells, the anterior endothelium, and next to this a homogeneous layer, both of which have crypts or openings leading into the interior of the iris tissue, thus placing its spaces in free communication with the cavity of the anterior chamber and allowing of rapid change in volume. The posterior surface is covered by the posterior limiting membrane and the pigment layer. The former contains very even tense fibres extending in a radial direction from the ciliary to the pupillary margin, and is regarded as a dilator pupillæ; as no muscular fibres have been demonstrated here, its tissue probably acts

by elastic traction. The pigment layer covering the posterior surface extends to the pupillary margin, and turns round to appear a little on the anterior surface, becoming easily visible where the lens is cataractous. (Figs. 6, 155, and 156.)

The color of the iris is determined by the amount of pigment, two kinds of which exist, the one lying in the branched cells of the stroma, and the other filling up the epithelial cells of the posterior pigment layer, pars iridica retinæ. With the exception of albinotic eyes (Fig. 155), the retinal layer always abounds in pigment, while that of the stroma varies, so that when the latter contains little pigment that of the epithelial cells shows through, the thin iris appearing blue. If the stroma be deficient in pigment but thick, the iris appears gray; and if there be a great amount of pigment in the stroma, brown, the depth of color varying with the amount. Isolated patches of pigment are found in the stroma as nævi or spots on a brown, gray, or blue iris. The pigment may be deeper in one part than another. The color of the iris changes in the early years of life, at first the stroma containing but little pigment and being very thin. With increasing age the stroma becomes thicker, and if the pigmentation does not increase, the iris becomes light blue or gray; if it increases, the color becomes brown.

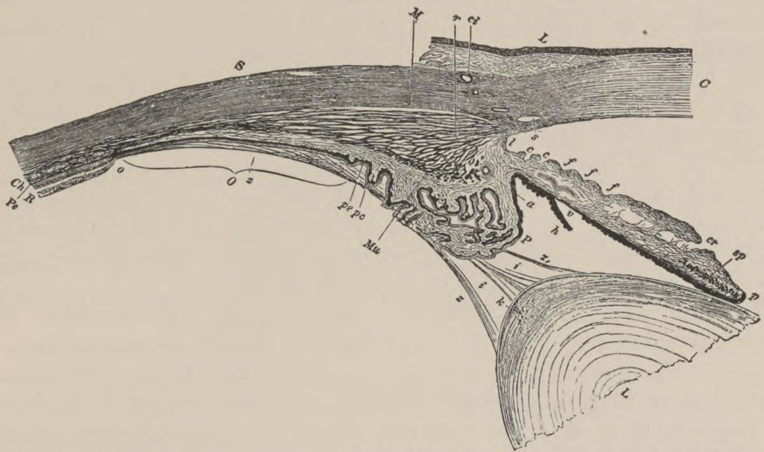
Ciliary Body. Macroscopic Anatomy. The ciliary body is the middle segment of the uvea, extending from the sclerocorneal juncture in front to the ora serrata behind. It is a circular organ, but when the eye is bisected the region appears as a triangle, the longer and outer side lying next to the sclera, the short anterior side against the pectinate ligament, and the inner margin in apposition with the pars ciliaris retinæ. It has three distinct subdivisions: the ciliary ring, the processes, and the muscle. The muscular portion is larger in hyperopic than in emmetropic eyes, and is smaller in myopic eyes.

Microscopic Anatomy. Proceeding from without inward, we find the ciliary muscle, which consists of an external portion containing the longitudinal or meridianal fibres which arise from the external tunic of the eye at the boundary between the cornea and sclera, and run straight backward until they are lost in the external layers of the choroid; the fibres here radiate and are transposed into circular fibres. (Fig. 156.) The ciliary processes (Plate XII., C, D) are a connective-tissue stroma containing a large number of bloodvessels and branched pigment cells placed upon the ciliary muscle. The layer next to the vitreous is a single stratum of non-pigmented cylindrical cells. Under this is a layer of pigmented cells, the pigmented epithelium; these two form the pars ciliaris retinæ. Under these is a homogeneous membrane, the hyaline lamella of the ciliary body.

The iris and ciliary body are attached to the sclera a little back of the corneoscleral margin by connective tissue, which is called the ligamentum pectinatum. (Fig. 157.) This forms an angle with the iris and cornea, forming the sinus of the anterior chamber, and where

attached to the sclera there is an annular lymph space forming the canal of Schlemm; this portion includes the ciliary ring.

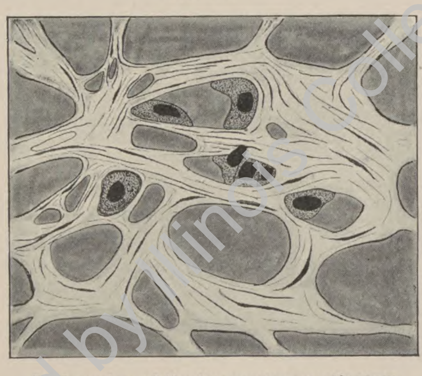
FIG. 156.



Meridianal section through anterior part of the eye, showing the ciliary body and iris, with neighboring structures. *C.* Cornea. *S.* Sclera. *s.* Schlemm's canal. *L.* Limbus conjunctivæ. *ci.* Anterior ciliary vein. *l.* Ligamentum pectinatum. *cr.* Crypts in circulus minor iridis. *c.* Periphery of iris. *f.* Contraction furrow. *hvp.* Retinal pigment of iris. *v.* Anterior layer of retinal pigment. *p.* Pupillary margin. *sp.* Cross-section of sphincter pupillæ. *M.* Longitudinal fibres of ciliary muscle, Brücke's portion. *Mu.* Circular fibres or Müller's portion. *r.* Transition or radial fibres. *a.* Circulus arteriosis iridis major. *P.* Ciliary processes. *pe.* Pigment cellular layer. *Pe.* Pigment epithelium. *pc.* Non-pigmented layer. *R.* Retina. *O.* Orbiculus ciliaris. *o.* Ora serrata. *ch.* Choroid. *z.* Fibres of zonule of Zinn. z_1. Free portion of zonula. *i.* Canal of Petit. *L.* Lens. *k.* Nuclei of lens. Magnified 14 times. (After FUCHS.)

The anterior chamber of the eye is formed in front by the cornea, behind by the iris, in the region of the pupil by the anterior capsule

FIG. 157.



Surface view of the ligamentum pectinatum.

of the lens, and at its margins by the ligamentum pectinatum, behind which lie the canal of Schlemm and the anterior region of the ciliary

body. The depth of the anterior chamber is influenced by accommodation, being shallower during the act from protrusion of the anterior surface of the lens; it is greatest in young persons and shallower in old age; myopic eyes have a deep anterior chamber, hyperopic eyes a shallow one. Where the tension of the eye is increased, the anterior chamber becomes shallower.

The posterior chamber is an annular space at the edge of the lens, being produced by the iris coming in contact only at its pupillary margin with the anterior capsule of the lens. It is bounded in front by the iris, to the outer side by the ciliary body, its inner and posterior wall being formed by the lens and the zonule of Zinn, the latter approaching from the inner space between the lens and the ciliary body. The two chambers communicate only by means of the pupil, and both are filled with the aqueous humor.

Choroid. **Macroscopic Anatomy.** On opening the eyeball and removing the vitreous and retina, the inner surface of the uvea is exposed; the choroid extends from the ora serrata to the optic nerve, appearing as a smooth brown membrane. On removing this from the underlying sclera, it is found to be attached more firmly at some spots than others, more particularly at the optic nerve, at the entrance of the ciliary arteries and nerves, and at the equator in the region of its large veins, the venæ vorticosæ. Thus the outer portion appears to be shaggy, on account of adherent shreds of membrane.

Microscopic Anatomy. The thickness of the choroid varies from 0.08 mm. at the optic aperture to 0.05 mm. at the ora serrata. It has five outer layers (Plate XII., E, F, G), being from without inward: (1) the suprachoroid, which is a richly pigmented layer of fibrous tissue; (2) the layer of large vessels, which are mainly veins, the intervascular spaces being richly supplied with pigment cells; (3) the layer of medium-sized vessels, which is but slightly pigmented; (4) the layer of capillaries, which is non-pigmented. These capillaries have a very wide bore and are packed closely together, with their interspaces narrower than the capillaries themselves; (5) the lamina vitrea, which is a homogeneous membrane lining the inner surface of the choroid. Upon this lies a single layer of cells which have been developed from the retinal mesoderm, which are deeply pigmented and belong to the retina, the pigmented epithelium of the retina.

The choroidal stroma consists of a ground substance of loosely interwoven connective-tissue lamellæ containing bloodvessels, white fibres, and elastic tissue with stellate pigmented cells.

Ophthalmoscopic Appearance of the Choroid. This membrane gives the characteristic color to the fundus, and the amount of pigment therein is responsible for much of the variations found in normal and diseased conditions. The pigment is contained: (1) in the pigment epithelium of the retina (Fig. 158); (2) in the stroma of the choroid (Fig. 159). If the pigment be wanting in both of these structures, we have the albinotic fundus (Plate XII., E), which is

light red, the entire larger circulation of the retina and choroid being visible; on account of the overlying capillary vascular layer of the choroid the intervascular spaces between the larger bloodvessels show as pink. Where the pigment is wanting entirely, or there is but little in the pigmented epithelial cells of the retina (Plate XII., F), while that of the choroidal stroma is more or less normal in amount, the tessellated fundus is observed, in which the intervascular spaces appear as dark plaques. Where the pigment epithelium and the stroma are heavily stained (Plate XII., G) the choroidal circulation is not visible, and the fundus is of a dark hue. This type of fundus exists in the dark races, varying from a dark brown in the Chinaman, Indian, and Malay, to a slaty hue in the negro. Occasional bizarre effects are seen, as in the fundus flavus. The ophthalmoscopic appearance of the normal average fundus lies between these

FIG. 158.

FIG. 159.

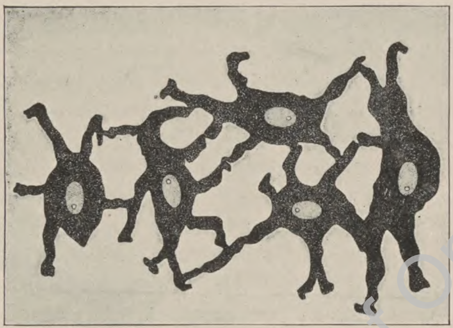


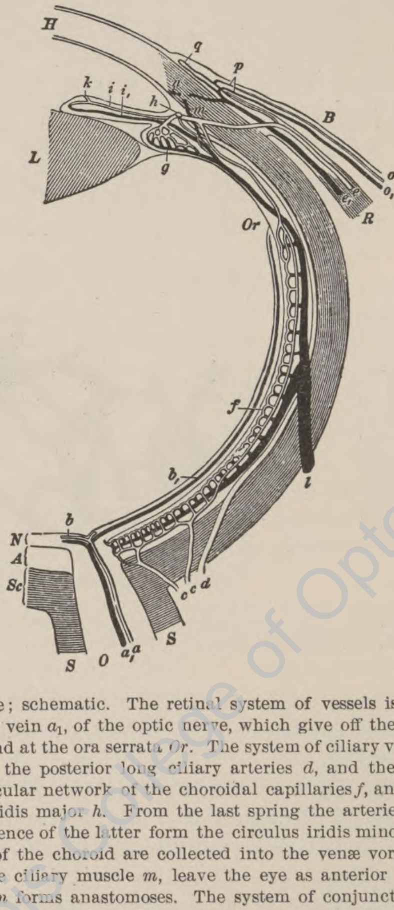
FIG. 158.—Hexagonal pigment cells of the retina.
FIG. 159.—Pigment stroma cells of the choroid.

extremes. In the blonde more of the choroidal circulation is observable than in the brunette, and in the latter the intervascular spaces are seen more distinctly.

Bloodvessels of the Eye. The bloodvessels of the eye belong for the most part to the uvea, which is made up for the greater part of vascular tissue, and, hence, is very liable to become inflamed. Fuchs describes the ocular vascular system as follows: Three systems of bloodvessels exist in the eye: that of the conjunctiva, that of the retina, and that of the uvea (ciliary system of vessels). The arteries of this system are: 1. The posterior ciliary arteries; these arise from the ophthalmic artery and enter the interior of the eye through the sclera in the region of the posterior pole. The majority of them pass at once into the choroid (short posterior ciliary arteries). (Fig. 160, *c*, *c*.) Two of them, however (the long posterior ciliary arteries) (Fig. 160, *d*), run, one on the outer side, the other on the inner side, between the choroid and sclera and as far forward as the ciliary muscle. Here each divides into two branches, which run in a direc-

tion concentric with the margin of the cornea, and unite with the branches of the artery of the opposite side to form an arterial circle, the circulus arteriosus iridis major. (Fig. 160, *h*, and Fig. 156, *a*.) This gives off the arteries for the iris, which extend radially from its ciliary to its pupillary margin. (Fig. 160, *i*.) Shortly before they

FIG. 160.



Bloodvessels of the eye; schematic. The retinal system of vessels is derived from the central artery *a*, and the central vein a_1, of the optic nerve, which give off the retinal arteries *b*, and the retinal veins b_1. These end at the ora serrata *Or*. The system of ciliary vessels is fed by the posterior short ciliary arteries *c c*, the posterior long ciliary arteries *d*, and the anterior ciliary arteries *e*. From these arise the vascular network of the choroidal capillaries *f*, and of the ciliary body *g*, and the circulus arteriosus iridis major *h*. From the last spring the arteries of the iris *i*, which at the smaller (inner) circumference of the latter form the circulus iridis minor *k*. The veins of the iris i_1, of the ciliary body and of the choroid are collected into the venæ vorticosæ *l*; those veins, however, that come from the ciliary muscle *m*, leave the eye as anterior ciliary veins e_1. With the latter, Schlemm's canal *n* forms anastomoses. The system of conjunctival vessels consists of the posterior conjunctival vessels *o* and *o*. These communicate with those branches of the anterior ciliary vessels which run to meet them—that is, with the anterior conjunctival vessels *p*—and form with these the marginal loops of the cornea *q*. *O*. Optic nerve. *S*. Its sheath. *Sc*. Sclera. *A*. Choroid. *N*. Retina. *L*. Lens. *H*. Cornea. *R*. Internal rectus. *B*. Conjunctiva (After LEBER, from FUCHS.)

reach the latter they form by anastomosis a second, smaller vascular circle, the circulus arteriosus iridis minor, or the small circle of the iris. (Fig. 160, *k*.) 2. The anterior ciliary arteries come from in front, arising from the arteries of the four recti muscles. (Fig. 160, *e*.) They perforate the sclera near the margin of the cornea, and assist

in forming the circulus arteriosus iridis major. The short posterior ciliary arteries are therefore designed mainly for the choroid, the long posterior ciliary arteries and the anterior ciliary arteries for the ciliary body and iris.

The arrangement of the veins is essentially different from that of the arteries. In the choroid the capillary network of the chorio-capillaris (Fig. 160, *f*) is fed by the arteries. The blood from this flows off through a great number of veins that unite to form larger and larger trunks. A number of these trunks simultaneously converge to a common centre, where, consequently, a sort of whorl or vortex is produced by veins coming together from all sides. These vortices, the number of which amounts to four at least, usually more, lie somewhat behind the equator of the eye; from them are given off the venæ vorticosæ, which, perforating the sclera in a very oblique direction, carry the blood to the outside. (Fig. 160, *l*.)

In the ciliary processes the arteries break up into a greater number of twigs, which pass over into thin-walled veins. (Fig. 160, *g*.) These constitute the greater part of the ciliary process, which, accordingly, consists mainly of vessels. The larger veins, which are formed by the union of these vessels, and also most of the veins of the ciliary muscle, pass backward to the venæ vorticosæ. The veins that come from the iris (Fig. 160, i_1) likewise pass to the venæ vorticosæ. Hence, almost all the venous blood of the uvea empties into the latter. A portion of the veins coming from the ciliary muscle (Fig. 160, *m*), however, take another course, as they pass out directly through the sclera, and thus come into view beneath the conjunctiva near the margin of the cornea (anterior ciliary veins, Fig. 160, e_1). In their course these correspond to the anterior ciliary arteries; they constitute principally the violet-colored vessels which are seen running backward beneath the conjunctiva in ciliary injection or in stasis within the eyeball (glaucoma). The anterior ciliary veins anastomose with the conjunctival veins and also with Schlemm's canal. The latter is a venous sinus running along the sclerocorneal junction. (Fig. 160, *n*, and Fig. 156, *s*.)

Nerves of the Uvea. The nerves of the iris are derived from the ciliary plexus. They are at first medullated and quickly reunite within the ciliary zone to form the iridian plexus, which becomes denser as it approaches the sphincter. Three kinds of fibres arise from this plexus: (1) non-medullated fibres belonging to the sympathetic pass backward toward the dilatator iridis; (2) medullated fibres, apparently sensitive, pass to the anterior surface; (3) medullated fibres pass to the sphincter and give it motor influence. Certain vasomotor fibres pass to the coats of the vessels. There are no ganglion cells in the iris. Its tactile sensibility is not great, and operations are not very painful if traction be avoided. Inflammation, however, is attended with great pain.

The *ciliary nerves* supply the ciliary muscle and processes. The long nerves are sensitive, being derived from the nasal branch of

the ophthalmic; the latter are from the ciliary ganglion, and are doubtless of a mixed character. The ciliary nerves penetrate the sclera near the optic disk, running forward in the suprachoroidal space, enter the ciliary muscle, and unite to form the ciliary plexus, which contains a few nerve cells. Fibres are given off from this plexus which pass to the cornea, iris, and ciliary muscle. These nerves end as follows: (1) vasomotor endings in the walls of the ciliary vessels; (2) motor endings in the ciliary muscle; (3) extremely fine reticulations of granular nerve fibres, which probably minister to ordinary sensation; (4) terminal arborescences, which are believed to have to do with the muscular sense which is particularly developed in the ciliary muscle. The sensory nerves of the ciliary body are abundant, and hence inflammation of this structure is attended with pain.

The *nerves of the choroid* are derived from twigs given off from the long and short ciliary nerves as they pass between the fibres and vascular tunics in their course to the ciliary body. The special branches destined for the choroid form a wide-meshed plexus of both medullated and non-medullated fibres within the lamina suprachoroidea. Ganglion cells, isolated or in limited groups, are found in this plexus and also along the vessels; the nervous supply of the choroid is distributed especially to the muscular tissue of the blood-vessels, and belongs to the vasomotor system. The choroid contains no sensory nerves, and inflammation of this membrane runs its course without pain.

Lymph Passages. There are no true lymph vessels in the eye, except in the conjunctiva; there are, however, large lymph channels and spaces (Fuchs):

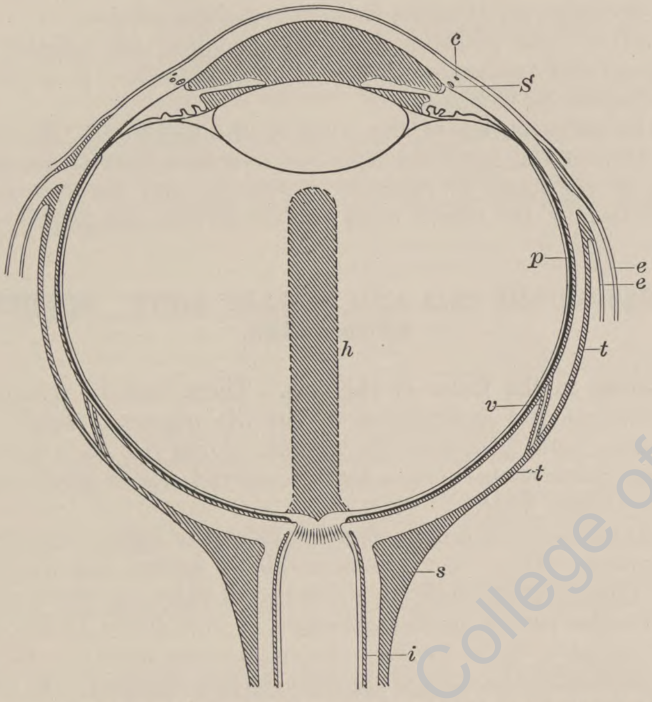
1. *Anterior Lymph Passages.* The lymph of the anterior section of the eye is collected into two large lymph spaces, namely, the anterior and posterior chambers, which communicate by means of the pupil. The outflow of lymph from these spaces takes place by its discharge from the posterior chamber through the pupil into the anterior chamber; thence it filters through the meshwork of the ligamentum pectinatum into the subjacent Schlemm's canal (Fig. 161, *s*), and from there gets into the anterior ciliary veins (*c*), with which Schlemm's canal is in direct communication.

2. *Posterior Lymph Passages* These are as follows: (*a*) The hyaloid canal, or central canal of the vitreous (Fig. 161, *h*), which extends from the point of entrance of the optic nerve forward as far as the posterior pole of the lens. During the development of the eye this canal lodges the hyaloid artery, which in the fully formed eye disappears, while the canal remains. It has its outlet in the lymph spaces of the optic nerve. (*b*) The perichoroidal space (Fig. 161, *p*) is the space between the choroid and sclera. It is continued along the vessels which pass through the sclera, especially the venæ vorticosæ (Fig. 161, *v*), and thus communicates with the anterior ciliary veins (Fig. 161, *c*), Tenon's space (Fig. 161, *t*, *t*), which lies between the sclera and Tenon's capsule. The outflow of lymph from all these

spaces takes place into the lymph passages which spread out along the optic nerve. These latter are (*D*) the intervaginal space, which is found between the sheaths of the optic nerve (Fig. 161, *i*), and (*E*) the supravaginal space (Fig. 161, *s*), which surrounds the sheaths of the optic nerve.

By far the greatest amount of lymph leaves the eye through the anterior lymph passages. These, therefore, are the more important. Their impermeability leads to serious changes in the eye (glaucoma), while up to the present time nothing certain is known in regard to disturbances of the function of the posterior lymph passages.

FIG. 161.



Lymph passages of the eye: schematic. *S.* Schlemm's canal. *c.* Anterior ciliary veins. *h.* Hyaloid canal. *p.* Perichoroidal space, which communicates by means of the venæ vorticosæ *v*, with Tenon's space *t*, *t*. *s.* Supravaginal space. *i.* Intervaginal space. *e e.* Continuation of Tenon's capsule upon the tendons of the ocular muscles, lateral invagination. (After FUCHS.)

Nutrition of the Eye. The nourishment of the eye comes mainly through the uveal vessels; the secretion of the fluids of the eye is also indirectly effected by the uvea. The aqueous humor is the only secretion of the eyeball proper. It is a limpid liquid containing a small amount of albumin, secreted mainly by the ciliary processes, being poured first into the posterior chamber, thence passing through the pupil into the anterior chamber, leaving the eye through Schlemm's canal and the ligamentum pectinatum. It is secreted and excreted

rapidly in health, and is restored quickly after evacuation of the anterior chamber by operation, sooner in youth than in old age. The fluid that accumulates in the anterior chamber after evacuation of the aqueous contains more albumin than the normal aqueous.

The cornea is nourished by the marginal loops of bloodvessels at the limbus, and somewhat by the aqueous humor which diffuses into its tissue. The lens and the vitreous obtain nourishment mainly from the ciliary body and the anterior section of the choroid; hence, in diseases of these structures the lens and vitreous become clouded, and may undergo degeneration. The internal layers of the retina are nourished by the retinal vessels, the outer layers being dependent upon the choroid; the regeneration of the visual purple is accomplished through nourishment from the choriocapillaris.

The intra-ocular pressure is dependent upon the relation of the capacity of the ocular envelopes to the contents. It is discussed in the chapter on Glaucoma.

For the participation of the uvea in the visual act, see page 65; the reaction of the pupil to light and accommodation, see page 30; reaction of the pupil to poisons, mydriatics, and myotics, see page 112; reaction of the ciliary body to cycloplegics, see page 112.

DISEASES OF THE IRIS AND CILIARY BODY. CONGENITAL ANOMALIES.

Variations in the Color of the Iris. There may be irregularities in the amount and distribution of the iris pigment, which may be massed into little heaps in the stroma, giving rise to a number of brown or black spots upon a lighter colored iris or patch upon its surface. (Plate XIII.)

Sometimes one iris differs in color from the other; this is called *heterochromia*. When one eye is decidedly brown and the other a uniform blue or gray, indicating absence of pigment, the latter may have been the site of previous disease, or it is liable to be affected later by cataract, while the dark eye may remain normal. In inflammatory conditions the color of the iris always is changed. In albinism the iris usually has a pink appearance, which is due to the shining of the fundus reflex through the iris stroma.

Membrana Pupillaris Perseverans. As has been noted in the chapter on the development of the eye, a vascular membrane fills the pupillary area which nourishes the lens. It is of comparatively frequent occurrence in newborn infants, but, as a rule, is resorbed, entirely before birth or shortly afterward. In a few cases complete resorption does not take place, and a gray or brown tissue lies upon the anterior capsule of the lens, arising from the circulus minor iridis; in the centre it is attached to a small round white capsular opacity. When of such a degree as shown in Plate XIII., Fig. 12, it interferes seriously with visual acuity. Many cases, however, display only

one or two filaments from one portion of the pupillary margin to the opacity, or from the iris to the capsule of the lens, or in other cases only a few brown dots remain upon the lens capsule. If the pupil be dilated by atropine, it opens fully, as the fibres are very extensible. It is otherwise the case in posterior synechiæ, for here the characteristic clover-leaf formation of the pupil is observed as it becomes enlarged. (Plate XIII., Fig. 9.)

DESCRIPTION OF PLATE XIII.

FIG. 1.—Myosis from eserine; direct illumination, showing the full extent of the markings of the iris; the pupillary portion stretched by contraction of the circular fibres; the pupil is never perfectly round, and is usually situated downward and inward.

FIG. 2.—Mydriasis from atropine; ophthalmoscopic illumination; fullest expansion of the pupil by contraction of the radial fibres; the distinctive markings on the iris are nearly obliterated; the lens star shows indistinctly.

FIG. 3.—Corectopia, displacement of the pupil; direct illumination. In this condition the pupil is usually somewhat irregular, small, and displaced to one side of the iris, usually downward and inward; in congenital forms being sometimes associated with coloboma of the choroid.

FIG. 4.—Glaucoma, the iris structure being ill defined from swelling; the pupil irregular, dilated, and having a greenish reflex.

FIG. 5.—Iridectomy for glaucoma; ophthalmoscopic illumination. The edges of the coloboma even and the whole pupil being the shape of an inverted keyhole; the upper edge of the lens and the ciliary processes are seen.

FIG. 6.—Imperfect healing in iridectomy for glaucoma; anterior synechia caused by incarceration of one edge of the coloboma in the corneal wound; direct illumination.

FIG. 7.—Optical iridectomy for leucoma of the cornea; direct illumination. The coloboma is usually made downward and inward on the back of the clearest portion of the cornea; small iridectomy which does not reach to the root of the iris.

FIG. 8.—Congenital coloboma of the cornea; direct illumination. When the coloboma reaches to the foot of the iris it is usually accompanied by coloboma of the choroid.

FIG. 9.—Iritis with posterior synechiæ; ophthalmoscopic illumination. The markings of the iris are not well defined; the synechiæ show dark, forming the characteristic clover-leaf pupil.

FIG. 10.—Exclusion of the pupil or total posterior synechiæ from chronic iritis; ophthalmoscopic illumination. The whole edge of the iris and sometimes the entire posterior surface of the iris are bound down by adhesions to the anterior capsule of the lens; this condition and the following (Fig. 11) are prone to give rise to secondary glaucoma.

FIG. 11.—Occlusion of the pupil from iridocyclitis; oblique illumination. The pupillary area is filled with organized exudation; the pupillary margin of the iris being bound down to the anterior capsule of the lens, the centre of the iris being bulged forward, causing the condition known as iris bombé.

FIG. 12.—Persisting pupillary membrane; direct illumination. A few strands reach from over the edge of the pupillary margin to the centre of the lens. This condition is frequently associated with persisting hyaloid artery.

FIG. 13.—Iridodialysis (ophthalmoscopic illumination), forming two pupils and associated with double vision. This condition and that of the next are of traumatic origin.

FIG. 14.—Polycoria; ophthalmoscopic illumination. In this patient there were three pupils and triple vision; the edge of the lens and ciliary processes could be distinctly seen.

FIG. 15.—Foreign body in iris and lens, binding the iris down to the lens; this being aseptic, was not associated with inflammatory changes, and was retained in the eye ten days before extraction by the magnet; but slight opacity of the lens capsule followed. Direct illumination.

FIG. 16.—Traumatic hemorrhagic iritis with hyphæmia simulating hypopyon. Direct illumination.

FIG. 17.—Anterior synechia with hernia of the iris from incarceration in corneal wound. Direct illumination.

FIG. 18.—Sarcoma of the iris; oblique illumination. This was attended by iritis and posterior synechia.

FIG. 19.—Syphilitic iritis; direct illumination. Gumma of the iris.

FIG. 20.—Acute choroiditis; direct illumination. In this condition the exudation in the vitreous gives rise to a yellowish-green reflex from the pupil.

Treatment. As a rule, there is little disturbance of vision; but if the membrane be very thick, the strands may be divided by the iridectomy scissors or the sharp hook.

Coloboma Iridis. Congenital coloboma of the iris is always situated below, the pupil, being continued downward to the margin of the cornea, growing continuously narrower, the sphincter lining the margin of the pupil and also the coloboma as far as its apex. (Plate XIII., Fig. 8.) In the majority of cases a small rim of iris may be observed at the bottom of the coloboma. It is to be distinguished from the one made by iridectomy; in the latter the sphincter is wanting in the coloboma, and it may be seen to end with sharp edges at the dividing line between the pupil and the coloboma. (Plate XIII., Figs. 5, 6, 7.) If the coloboma be accompanied by a slight defect of the choroid, there may likewise exist a deficiency in the ciliary body; sometimes coloboma or indentation in the edge of the lens accompanies. There are no special symptoms and no treatment.

Aniridia vel Irideræmia. The iris may be entirely absent or but a small residual portion remain; this defect is accompanied generally by congenital opacities in the lens and cornea and other abnormalities. For the special condition, dark glasses or the stenopœic hole may be indicated.

Ectopia Pupillæ vel Corectopia. Normally the pupil is not precisely in the centre, but is usually a little below and to the side. This displacement is sometimes so great that it is noticeable, especially in myosis. (Plate XIII., Fig. 1.) It may even be situated eccentrically in the neighborhood of the corneal margin (Plate XIII., Fig. 3), and is sometimes complicated with dislocation of the lens.

Policoria or multiple pupils have been described as occurring congenitally, but as a rule such conditions are due to traumatism. (Plate XIII., Figs. 13, 14.)

Inflammatory Diseases. Inflammation of the iris is intimately connected with that of the ciliary body as both are supplied by the same bloodvessels, and the iris springs directly from the ciliary body, forming a continuous tissue. It should likewise be remembered that the choroid is a portion of the uvea, and is usually more or less affected by inflammations of the anterior portions, particularly where the ciliary body is involved. Thus, while we speak of an iritis, a cyclitis, or a choroiditis, an inflammation of these tissues is more properly a uveitis. The disease may, however, be so predominant in the iris or ciliary body that the affection may be classed as iritis or cyclitis, and for practical purposes the symptoms of the predominant lesions may be described separately.

Hyperæmia of the Iris. Congestion is the first stage of inflammation, and either may go on to resolution or to later stages of inflammation. Every iritis is preceded by hyperæmia, but cases are met with that do not proceed further than this stage. It may also be coincident with or be a symptom of inflammation in other portions of the uveal tract or the neighboring ocular tissues; thus it accom-

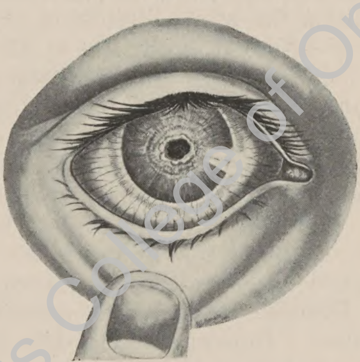
panies inflammatory changes in the ciliary body and acute, severe choroiditis, also ulcer of the cornea and scleritis.

Hyperæmia of the iris is characterized by change in its color, so that a blue or gray iris becomes greenish and a brown iris yellowish red; in dark eyes the discoloration is not so marked as in blonde eyes. One of the first symptoms is pericorneal congestion, a characteristic of affections of the uvea and cornea, consisting of enlargement of the fine vessels situated in the episcleral tissue radiating from the corneal margin. (Fig. 162.) The pupil is sluggish and does not respond freely or quickly to light, accommodation, or mydriatics; as there is no exudate, posterior synechiæ do not form. In acute cases complete resolution takes place as the cause ceases. In chronic hyperæmia the iris becomes discolored from changes in the pigment cells, and the pigment at the pupillary border disappears, the edge becoming ragged and notched. Senile changes in the iris cause bleaching of a similar nature.

Hyperæmia of the Ciliary Body. Simple hyperæmia of the ciliary body is accompanied usually by changes in the choroid of a congestive or inflammatory type. The ciliary region becomes easily congested by use of the eyes or by irritation, and there is ciliary pain following close work.

Etiology. Hyperæmia being the first stage of inflammation, the cause of congestion of the iris or ciliary body may be looked for in eyestrain, injuries and inflammations of the cornea, sclera, choroid, and in disturbances of general nutrition.

FIG. 162.



Iritis. The pupil is irregularly contracted, and circumcorneal congestion is marked.

Treatment. Rest, dark glasses, instillation of atropine, removal of the general or local cause of the congestion, regulation of eyework, and correction of refractive errors.

Iritis. In addition to the symptoms of hyperæmia, true iritis is attended by exudation into the stroma of the iris and the anterior and posterior chambers.

1. *Exudation into the stroma of the iris* is attended by infiltration with round cells which thickens and swells the membrane. (Plate XII., B.) The discoloration is more pronounced than in hyperæmia, the distinctness of the markings on the anterior surface becoming obscured; the rigid and swollen iris reacts but little to light and accommodation, the pupil being greatly contracted. (Fig. 162.)

2. *Exudation into the anterior chamber* is manifested by turbidity of the aqueous from suspension therein of cells; the pupil looks gray instead of black; the exudate floating in the aqueous sinks into

the bottom of the chamber, producing hypopyon. With great hyperæmia, exudation of blood may take place, which sinks to the bottom of the anterior chamber—hyphæmia. (Plate XIII., Fig. 16.) There is considerable exudate upon the surfaces of the iris (Plate XII., B) and upon the walls of the anterior chamber; hence the cornea and lens appear cloudy on account of the deposit of numerous round cells upon their endothelium. These may even coalesce and be deposited in spots, but this condition is more common where the ciliary body is involved. If these exudates become organized, a membrane is formed, connected with the pupillary margin, which closes the pupil, causing the condition called *occlusion of the pupil* (Plate XIII., Fig. 11); this results in great impairment of vision.

3. *Exudation into the posterior chamber* cannot be seen directly on account of the iris being closely applied to the capsule of the lens; it gums down the iris, forming adhesions at the pupillary margin, or *posterior synechiæ.* (Plate XIII., Figs. 9, 10.) It is the layer of retinal pigment that becomes adherent, and as this deposit is formed when the iritis is at its height and the pupil contracted, when the pupil tends to resume its normal size, or if atropine be instilled, it is found that the iris retracts strongly at its unattached portions, forming cloverleaf adhesions. The tags jutting into the pupil appear dark brown or black, and there are isolated spots on the lens capsule, showing where the retinal pigment has been attached and torn away. Dilatation of the pupil by atropine at this time may release some or all of the adhesions, but the pigment remains permanently, giving evidence during the whole lifetime of the patient that iritis once existed.

If adhesion of the iris to the capsule of the lens exist around the whole extent of the pupillary margin, it is called *annular posterior synechia;* this results in shutting off the anterior from the posterior chamber, *exclusion of the pupil* (Plate XIII., Fig. 10), which does not of itself necessarily affect the sight if the pupil be free from membrane, but subsequently causes increase of tension and glaucoma, resulting in blindness. This condition frequently is associated with occlusion of the pupil, and as the latter rarely occurs without closure of the anterior and posterior chambers, it is subject to the same dangers.

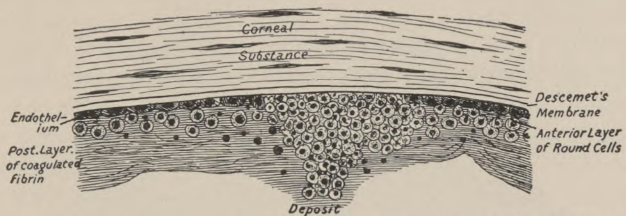
Cyclitis. Most writers describe cyclitis under the name of "serous iritis." Inflammation of the ciliary body is attended always with hyperæmia or with inflammation in the iris. Simple cyclitis, with but little inflammation of or even with but slight hyperæmia in the iris, may occur in a chronic form, the inflammatory symptoms being slight, the pupils generally somewhat dilated, the chief symptom of obscuration of vision being due to the presence of deposits on the posterior surface of the cornea (Fig. 164), and opacities in the vitreous.

Severe cyclitis may occur without marked symptoms being set up in the iridic tissue, which only becomes hyperæmic. Exudation into the anterior chamber is not usually pronounced, and while the iris reacts slowly to light, accommodation, and mydriatics, synechiæ do not tend to form.

Exudation from the ciliary body takes place into the anterior and posterior chambers and into the vitreous.

1. *Exudation into the anterior chamber* passes either directly from the anterior portion of the ciliary body through the ligamentum pectinatum at the sinus of the anterior chamber, or, being deposited directly in the posterior chamber, is carried with the aqueous through the pupil into the anterior chamber. In consequence of this, especially in the chronic forms of inflammation, conglomerations of cells aggluti-

FIG. 163.



Deposit upon posterior surface of cornea in cyclitis. The endothelium is intact except where the deposit is thickest. (After FUCHS.)

nated into masses by fibrinous exudate (Fig. 163) are thrown against the posterior surface of the cornea by the centrifugal force of the eye movements, and adhere in a triangular shape to the endothelium, the larger exudates being at the bottom, while the smaller are at the upper portion of the triangle. (Fig. 164, *A* and *B*.) These deposits are light gray or brownish, varying from a very small size to that of a pin's head, and formerly were supposed to be located in Descemet's layer (descemitis), but are readily distinguishable from macular deposits

FIG. 164.

Deposits on posterior surface of cornea in cyclitis. *A*. Larger deposits. *B*. Smaller.

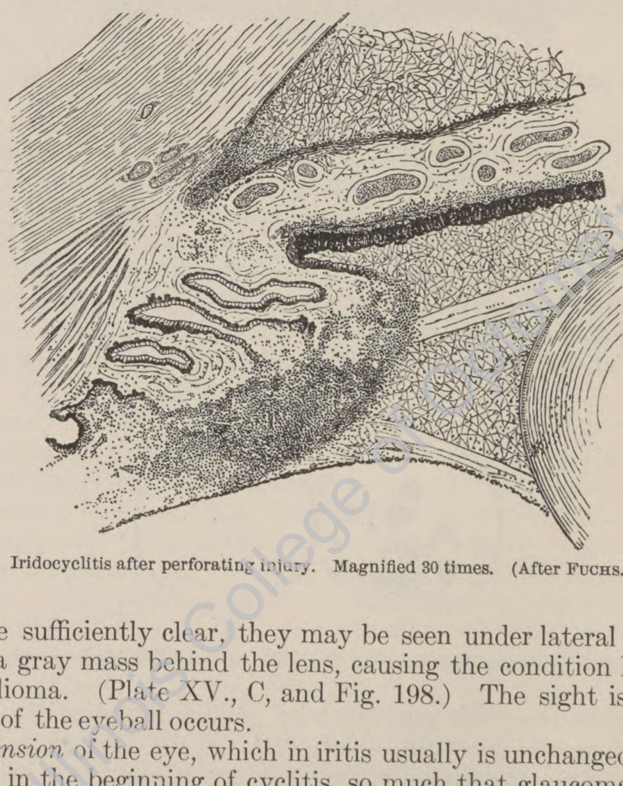
in the cornea (keratitis punctata) by oblique illumination, by their clear outline and brownish color, and by the fact that they are all on the same plane on the posterior surface, and not in different depths of the cornea. If the cornea be incised and the aqueous allowed to escape, some of the deposits are carried away. Pigment deposit on the surface of the lens as well as the posterior surface of the cornea has been seen following or during the course of cyclitis. If the exudate from the ciliary body into the anterior chamber be very great, it may be deposited in the form of hypopyon; but if this

occurs, grayish spongy masses will be found projecting around the angles of the anterior chamber.

2. The greater mass of exudate in cyclitis is deposited in the *posterior chamber*, and if extensive leads to adhesion of the whole posterior surface of the iris to the capsule of the lens—*total posterior synechia.* As it shrinks, this exudation draws the iris everywhere to the anterior surface of the lens, so that the posterior chamber is obliterated and the anterior chamber becomes proportionately deeper, especially at the periphery, where the iris is displaced farther backward. (Fig. 165.) This occasions the same danger of glaucoma as spoken of in exclusion of the pupil.

3. *Exudation into the vitreous* causes opacity, which if of large size and in the anterior portion causes great diminution of vision. If the

FIG. 165.



Iridocyclitis after perforating injury. Magnified 30 times. (After FUCHS.)

media be sufficiently clear, they may be seen under lateral illumination as a gray mass behind the lens, causing the condition known as pseudoglioma. (Plate XV., C, and Fig. 198.) The sight is lost and atrophy of the eyeball occurs.

The *tension* of the eye, which in iritis usually is unchanged, often is elevated in the beginning of cyclitis, so much that glaucoma may set in and blindness speedily be produced. In the later stages of cyclitis, on account of the shrinking of the exudates, diminution of the ocular pressure is more common.

The *subjective symptoms* of both iritis and cyclitis are those of severe inflammation, consisting of lacrymation, photophobia, and severe pain. The pain and tenderness are situated not only in the eyeball, but

also in the surrounding parts, especially the region of the eyebrows. In the acute cases the pain is intense, while chronic cases occur in which inflammatory symptoms are almost entirely wanting. In severe iridocyclitis the pain is intolerable, particularly at night, and is accompanied by hyperpyrexia and sometimes vomiting. Vision is always more or less diminished. On account of the increased refractive index of the aqueous in iritis, a pseudomyopia is developed in the course of the disease, which disappears after resolution takes place.

The following signs show *positive evidence of involvement of the ciliary body:* 1. When the inflammatory symptoms are very severe, especially if associated with œdema of the upper lid. 2. When the ciliary region is painful. 3. When deposits occur on the cornea. 4. When the anterior chamber becomes very deep from gumming down of the ciliary margin of the iris. 5. When the visual acuity is greatly lowered, which is due to involvement of the vitreous. 6. When the tension either is lowered or elevated.

Course and Sequelæ of Iritis and Cyclitis. **Course** Acute cases associated with marked inflammation run a severe course, the average case of iritis lasting from one to two months, the first signs of improvement being decrease of the congestion and pain and prompt action of atropine. Chronic cases show but slight symptoms of inflammation: an iridocyclitis or iridochoroiditis may last a number of years. Relapses of inflammation in the iris and ciliary body are common, being due to renewal of the exciting cause rather than to the mechanical effect of the adhesions which may have formed. Formerly it was supposed that posterior synechiæ were particularly dangerous, and many operations were devised for cutting the iris loose at its periphery. Unless the adhesions have caused exclusion or occlusion of the pupil, they should be left alone.

Complete resolution may take place in mild cases, if seen sufficiently early and the pupil kept dilated by atropine. Even if posterior adhesion of the iris has taken place, the pupil may fully dilate, leaving, however, pigment spots upon the anterior capsule of the lens, which do not become absorbed and may later be seen during the entire lifetime of the patient by oblique illumination or the ophthalmoscope; they are likewise subjectively evident as floating specks before the eyes. Hypopyon, hyphæmia, exudates into the anterior chamber, and slight opacities of the vitreous may disappear completely by resorption.

Sequelæ. In most cases permanent sequelæ remain after iritis and cyclitis: 1. The most common sequelæ of iritis are *posterior synechiæ;* these are evident by circumscribed adhesions of the iridic pigment at the pupillary margin to the anterior capsule of the lens, the pupil being irregular and responding to mydriatics incompletely in a clover-leaf form. (Plate XIII., Fig. 9.) Complete adhesion of the pupillary margin causes exclusion of the pupil, the body of the iris being pushed forward, producing the condition known as *iris bombé* (Plate XIII., Figs. 10, 11); the pupil being represented as a crater; the iris being greatly stretched becomes atrophic, elevation of intra-

ocular pressure occurs, and the symptoms of secondary glaucoma set in. On account of the increase of tension, the sclera may give way in places, forming ectasiæ. 2. *Atrophy of the iris*, especially of its pigment, appears as the result of repeated recurrences or chronic inflammation; the delicate markings of the anterior surface disappear, the pupillary margin is thinned down, and dilated vessels may often be distinguished. The iris pigment, particularly of the retinal layer, becomes absorbed and a black ring at the edge of the pupil is no longer seen, the edge of the iris looking frayed and its tissue lighter in color. The atrophic iris is very friable and makes performance of iridectomy very difficult. 3. *Occlusio pupillæ* is caused by organization of the exudate forming a pupillary membrane which diminishes the vision in proportion to its thickness. 4. *Exudates behind the iris :* total posterior synechiæ has been described. In severe cases the fibrous mass completely envelops the lens, and, as resolution goes on, has a tendency to shrink, causing the anterior chamber to become deeper from shrinking of the vitreous, and direct contraction causing detachment of the vitreous and retina. 5. *Atrophy of the eyeball* follows, which, on account of the diminished tension from the effect of the extra-ocular muscles pulling upon the ball, becomes of a quadrangular shape, being grooved at the insertion of the recti. The cornea becomes smaller, opaque, and flattened, at times remaining transparent, and becoming abnormally protuberant or thrown into folds. The lens and remaining vitreous become opaque and the eye blind. The eye becomes sensitive to touch, and secondary attacks of pain occur, especially if the eye harbors a foreign body, or deposits of bone or calcareous tissue develop. The atrophy pursues a course of months or years, and pain usually disappears when complete shrinkage occurs; the condition then is known as *phthisis bulbi*. 6. *Opacity of the lens* occurs on account of disturbed nutrition, particularly in cyclitis, as the iris and ciliary body become attached by exudates to the lens. Such a cataract is known by the name *cataracta accreta*. In atrophic eyeballs the lens is always opaque and shrunken.

Etiology of Iritis and Cyclitis. Iritis and cyclitis may arise as a primary process, the original site of the inflammation being in the iris or ciliary body. It is caused in the majority of cases by dyscrasiæ and *general* diseases. In such cases both eyes usually are affected, although not always at the same time. The inflammation may likewise arise as a *local* affection, under which heading we put traumatism and those idiopathic cases in which we can discover no definable cause; here the disease usually affects but one eye. To this subdivision belongs also sympathetic inflammation. Iritis and cyclitis may likewise arise as *secondary* affections from inflammation transmitted from the neighboring structures. In classifying inflammations of the iris and ciliary body according to the above scheme, we call them iritis or cyclitis or iridocyclitis according to the structure principally involved.

Primary Iritis and Cyclitis. *Syphilitic Iritis.* Syphilis is responsible for at least one-half of the cases of iritis. It is an early secondary symptom, appearing shortly after the first macular eruption, and occurs in 5 per cent. of the cases of syphilis. The inflammation has a characteristic appearance in that nodules of a yellowish-red color, of the size of a pin's head or larger, form either on the ciliary or pupillary margin of the iris, but never between. In the majority of cases these nodules disappear, leaving broad and solid synechiæ and atrophy of the iris tissue. In some cases no distinct nodules are formed, but the pupillary margin is swollen in places, and unusually broad synechiæ form which do not yield to atropine. Iritis may occur in the later stages of syphilis without the formation of nodules, but gummata (iritis gummatosa) (Plate XIII., Fig. 19) may develop in the iris and ciliary body, and, attaining great dimensions, break through the envelopes of the eye, bringing about its destruction.

Iritis likewise occurs in *hereditary syphilis*, although not so frequently, being usually associated with interstitial keratitis, occurring early in childhood, while acquired syphilis usually is observed in adults.

Inflammation of the choroid is associated with more than half of the cases of syphilitic iritis and cyclitis; the retina and optic nerve are frequently involved. There is a tendency to recurrence. The actual diagnosis can be established only by demonstration of the presence of syphilis or the favorable action of antisyphilitic remedies.

Iritis Scrofulosa. Iritis scrofulosa bears a resemblance to the iritis of hereditary syphilis, occurs in anæmic and scrofulous children and youths, and often is characterized by lardaceous-looking deposits which appear to come from the angle of the anterior chamber.

Iritis Tuberculosa. Tubercular deposits may occur primarily in the iris and ciliary body from wound infection, or, secondarily, in connection with general disease. They may take the form of miliary growths, with consequent inflammation, or may form large tumors. (Figs. 187 and 188.)

Iritis Rheumatica. Iritis rheumatica appears in persons of the rheumatic, arthritic, or uric-acid diathesis, is characterized by inflammation with little exudation, and has a marked tendency to recur.

Iritis Gonorrhœica. Iritis gonorrhœica develops where general infection has arisen from gonorrhœa. It is associated usually with gonorrhœal rheumatism, arising after the outbreak in the larger joints. It exhibits recurrences frequently associated with renewal of the urethral discharge or of the joint-affection.

Iritis has been seen in *relapsing fever* and *variola*. Iritis occurs in *diabetics*, associated with hypopyon.

Iritis Idiopathica. Idiopathic iritis is the form in which the cause remains obscure, being usually attributed to cold. The acute form is generally unilateral; the chronic form generally appears with symptoms of cyclitis and choroiditis, with light inflammatory symptoms, and runs a long course. This has already been described under the

heading Cyclitis. It has been called iritis serosa. When it occurs in persons of advanced age the cause seems to be defective nutrition, and it slowly progresses until blindness sets in.

Iritis Traumatica. The causes of traumatism of all kinds, especially perforation of the eyeball, traumatic iritis, and iridocyclitis, are described under their respective headings.

Iridocyclitis Sympathetica. Sympathetic inflammation is discussed on page 390.

Secondary Iritis and Iridocyclitis. Inflammation of the iris and the ciliary body may develop by transmission from neighboring structures, more especially suppurative keratitis and the deeper forms of scleritis; more rarely it is caused by severe conjunctivitis. Inflammations sometimes pass forward from the posterior section of the eye, from choroiditis, intra-ocular tumors, cysticercus, and swelling of the lens. Cysticercus and filaria have been observed in the anterior chamber and iris by Continental writers, and have been successfully removed.

Treatment of Iritis and Cyclitis. Most cases of iritis and cyclitis demand both local and general treatment.

LOCAL MEASURES. 1. The *mode of life requires modification;* physical exertion should be avoided, and, in severe cases, rest in bed is imperative. Because light excites the pupil to contraction, and on account of photophobia, *both eyes should be protected* by the patient being made to wear dark glasses and an eye-shade, and, as a rule, being kept in a moderately darkened room. (This is one of the few eye diseases in which a dark room is demanded. Confinement of eye patients to the necessarily poorly ventilated darkened room frequently does more harm, as regards recuperation, than the good that may be obtained from the absence of irritation from light; thus the dark room, except for the treatment for iritis and some cases of conjunctivitis, has almost disappeared from modern ophthalmic practice.) The *healthy eye should not be strained* by reading, and, as a rule, it should be put in a splint by the use of atropine, as the synergetic action of the pupils to light and accommodation is deleterious.

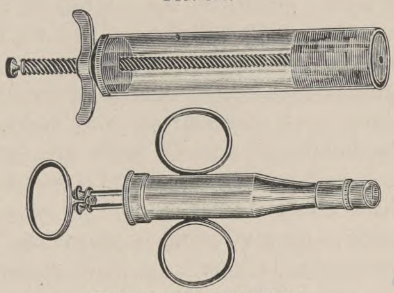
2. With the exception of traumatic cases (when during the first twenty-four hours *iced applications* may be used), *hot compressing* is indicated in all cases of iritis and cyclitis. The moist heat gives relief from pain and favors metabolism, thus hastening recovery. Moist heat may be applied by cloths wrung out of hot water, over which flannel may be laid to conserve the heat, the compresses being changed every two minutes. Several ingenious forms of applying heat by siphon or electric apparatus, under which moist cloths are kept, may be used.

3 *Extensive blood-letting* by the Heurteloup artificial leech (Fig. 166) applied to the temple once or twice, and repeated later, if necessary, may greatly diminish the inflammatory symptoms. Frequently after such a procedure the pupil yields for the first time

to the action of atropine. Natural leeches may be used, of which six or eight may be applied, but they are often unobtainable, are difficult of application, and are disgusting to the patient.

MEDICINAL TREATMENT. *Atropine* is the most important remedy in *iritis*, as it dilates the pupil, diminishes the amount of blood in the vessels, and counteracts the hyperæmia; by paralyzing the sphincter it puts the inflamed organ at rest; by enlarging the pupil it ruptures recent posterior synechiæ, as when the iris is fully contracted its edge is not against the lens, and it prevents the formation of adhesions. The amount of atropine used should be carefully regulated according to the intensity of the inflammation. As during the period of increase of inflammation, spasm of the sphincter exists, it is usually difficult to dilate the pupil, and atropine should be used in strong solutions (1 per cent. to 5 per cent. every three or four hours; or, if the pupil does not dilate, place a granule of atropine in the conjunctival sac, taking care to close the lacrymal puncta for a few minutes by stretching the skin over them with the finger-tip). By the simultaneous employment of cocaine (3 per cent.) or holocaine (1 per cent.), the action of atropine may be heightened. On account of systemic symptoms, strong solutions cannot be used more than a few times in succession, and may have to be combated by the administration of morphine internally. Atropine catarrh, from the continuous instillation of the drug, may occur; and if so, the mydriatic should be changed to scopolamine (0.1 per cent. to 0.5 per cent.) or duboisine (1 per cent.). If the inflammation be very severe, instillation of 1 : 1000 adrenalin chloride tends to reduce the congestion, not only in the external membranes of the eye, but also in the iris and ciliary body. Dionin, in 10 per cent. solution, instilled several times a day, is of marked value as a lasting local anæsthetic in cases of iritis as well as corneal ulcer.

FIG. 166.



Heurteloup artificial leech.

In cases of *iridocyclitis* in which the implication of the ciliary body is particularly prominent, and also in pure cyclitis, atropine is not well borne. If pain is caused by its instillation, or there is elevation of tension, atropine should be stopped, and dionin and adrenalin chloride alone used.

GENERAL MEASURES. 1. In all cases it is important to keep the alimentary tract in order by *regulation of the diet*, which should be limited to simple nourishing food, and *constipation combated*, preferably by saline cathartics.

2. The etiological factors should be considered, the majority of cases demanding general medical treatment. *Syphilitic iritis* offers

the most favorable prognosis, as it generally responds to energetic treatment. As the remedy should act promptly, mercury is given in the form of inunction (4 gm. of blue ointment or of the oleate rubbed into the arms and thighs twice a day, or the same quantity smeared on the soles of the feet), and is continued until the diseased eye is no longer inflamed, or until symptoms of ptyalism occur, and then iodide of potassium or sodium is administered in gradually increasing doses (1 to 10 gm., three times daily). The other alteratives, especially arsenic and gold, either singly or in combination with mercury, are of benefit (auri, arseni. et hydrargyri bromidi, āā 0.001 to 0.002 gm., three times daily).

The general treatment after the iritis has passed away is that laid down for syphilis in general.

In *hereditary syphilis* the treatment should be tonic as well as specific: syrup of the iodide of iron (1 to 2 c.c., three times daily), together with cod-liver oil (4 to 16 c.c., three times daily), syrup of hydriodic acid (2 to 4 c.c., three times daily). If gummata form and are not relieved by specific remedies, perforation of the eye may take place, and enucleation may have to be done. Other operations in the height of specific iritis are usually contraindicated.

In *iritis rheumatica* and *gonorrhœica*, sodium salicylate (1 to 2 gm., three times daily) or the oil of gaultheria (0.3 to 0.6 c.c.) are indicated, and give relief in about the same proportion of cases as in other rheumatic lesions. In *iritis diabetica*, arsenic seems to be of service. In *gouty* subjects the causes of the defective uric-acid elimination and such lesions as iritis are frequently assisted to disappear by cystogen or urotropin (0.3 to 0.5 gm., three times daily between meals), together with appropriate diet and lithia waters.

Treatment of the Sequelæ of Iritis and Iridocyclitis. Narrow and isolated *posterior synechiæ* may often be ruptured by the employment of atropine (1 to 5 per cent.) by itself, or in combination with holocaine (1 per cent.) or cocaine (5 per cent.). As a very energetic action is desired, it is secured most certainly by placing the pure drug direct in the conjunctival sac; the alternate use of myotics and mydriatics, the pupil being first contracted with eserine (0.2 per cent.), and then energetically dilated with atropine, is even more effective, but such procedures should not be made until some time after the iritis has been subdued. Broad synechiæ cannot be divided by such means, and were formerly operated upon (corelysis), as it was believed that the action of the iris in the opening and closing of the pupil, caused irritation which set up recurrence of the iritis; but it is now recognized that in such cases the original causal factor again acted, and, hence, such operations are now seldom practised.

Annular posterior synechia, with exclusio pupillæ in addition, demands iridectomy in order to restore communication between the anterior and posterior chambers, for, if allowed to remain, secondary glaucoma surely sets in. The operation is often difficult, on account of the shallowness of the chambers, due to protrusion of the iris

(*iris bombé*, Plate XIII., Figs. 10, 11), and also on account of atrophy of the tissue. Thus a good-looking coloboma is seldom the result in such cases, and we must be contented if a permanent opening remains, for the anterior chamber, in consequence of restoration of the connection between the two chambers, regains its normal depth, and a secondary operation may be performed later.

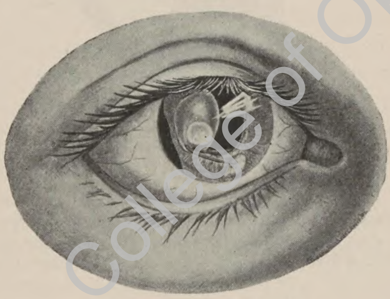
Total posterior synechia also requires iridectomy, which is frequently unsuccessful, as, on account of the adhesion of the iris by its whole posterior surface to the lens it is frequently impossible to excise a sufficiently large segment. In such cases the lens may be removed, together with more or less of the iris, or, if absent, iridotomy is indicated.

Neoplasmata of the Iris and Ciliary Body. *Benign Tumors. a. Cysts of the Iris. Serous cysts* may develop within the stroma of the iris after penetrating wounds of the eyeball, growing gradually until they reach the posterior surface of the cornea, and fill all or a portion of the anterior chamber. Their walls are formed by thinned iris tissue. They produce elevation of tension, and from this glaucoma results.

b. Dermoid tumors have been reported (Fig. 167), being caused by penetrating injuries by which epithelial cells are carried within, starting up tumor growth. These may even grow one or two hairs, as is the case with dermoids elsewhere.

Treatment. As such tumors are not benign when arising within the eye, they should be removed early by operation, by incising the cornea at a point corresponding with the growth, entering the forceps, withdrawing, and cutting out the tumor with the adjoining iris.

Fig. 167.



Dermoid tumor of the iris.

Melanomata are rare forms of real benign growths arising from proliferation of the pigment stroma cells growing into the anterior chamber. They rarely become of consequence, and are to be distinguished from sarcomatous growths by their slow course, small size, and absence of inflammatory symptoms. The pigment cells of the retinal layer at its reflection upon the anterior surface of the iris at the edge of the pupil may proliferate and project into the pupil; they sometimes become separated from the pupillary margin, dropping into the anterior chamber, but they rarely become larger than a grain of wheat, and are of very slow growth.

Malignant Tumors. Sarcomata of the iris may arise alone or in connection with the ciliary body. (Plate XIII., Fig. 18.) Those of the iris grow very slowly at first, appearing as pigmented brown tumors, finally filling the anterior chamber; they then grow back into the ciliary

region and fill the interior of the eye, breaking through the ocular envelopes, and extending their growth externally. Sarcomata of the ciliary body, like those of the iris and choroid, at first give rise to but little inconvenience, but, finally, from their size, cause pressure symptoms, following the same course as those of the iris and choroid.

These are more particularly described in the chapter on Neoplasmata of the Choroid.

Treatment. Malignant growths of the iris when very small may perhaps be excised, but it is safer for the life of the patient where they are of any size, and in those involving the ciliary body, to remove the eyeball as soon as the diagnosis is made.

Tubercular tumors usually occur in children and young adults. They generally begin as a tubercular iritis, but when the nodules

FIG. 168.

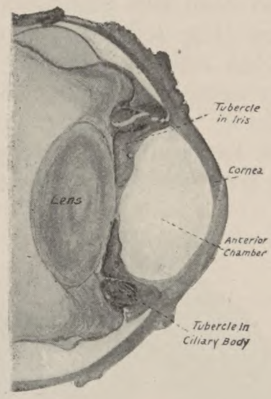


FIG. 169.

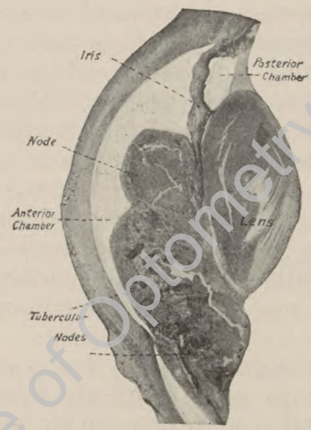


FIG. 168.—Tuberculosis of ciliary body and iris causing cyclitis, showing occlusion of pupil with retraction of iris and deep anterior chamber. Total posterior synechia. (Photograph from specimen.)

FIG. 169.—Tubercular tumor of iris, anterior chamber shallow from being almost filled by neoplasm, which fills lower part of posterior chamber. (Photograph from specimen.)

enlarge and form tumors, their course is much the same as that of malignant growths, and they are amenable to the same treatment—*i. e.*, enucleation.

Gummata of the iris and ciliary body form as yellowish tumors of somewhat rapid growth, occurring during the course of acquired or inherited syphilis as one of the later manifestations. (Plate XIII., Fig. 19.) They seem to be rapidly resorbed on exhibition of mercurial inunctions and large doses of iodide of potassium.

Very rare forms of iridic tumors, such as vasculomata, myomata, and myosarcomata from the ciliary muscle, carcinoma from the pars ciliaris retinæ, and lepra nodules, have been reported.

Injuries of the Iris and Ciliary Body. *Wounds and Foreign Bodies. Penetrating wounds of the iris* are complicated by wounds of the cornea, and usually with that of the lens and ciliary body. If the ciliary body be injured, the conjunctiva, the sclera, choroid, retina, and vitreous, as well as the iris, are usually affected. If the penetrating body be clean, infection does not take place, there is usually but little bleeding, and healing results, with damage dependent upon the extent of the injury. If a wound of the iris be accompanied by infection, inflammation results which may extend to the ciliary body and choroid, with resultant iritis and iridochoroiditis. Sympathetic ophthalmitis may arise from infected iris injuries, but is usually due to those of the ciliary region.

Foreign Bodies in the Iris. Non-infectious foreign bodies may remain in the anterior chamber, or be encapsulated in the tissue of the iris for a long time without giving rise to inflammation. They may be removed by corneal incision and the iris forceps, with or without section being made of the iris. (Plate XIII., Fig. 15.) The extraction of pieces of steel or iron from the anterior chamber by the electromagnet offers the most promising prognosis for the operation. (These subjects are more specifically described in the chapter on Sympathetic Inflammation.)

Traumatic Changes in the Iris. *Lacerations of the iris* usually start from the pupil, and may extend to the ciliary margin, so that the pupil appears to be pear-shaped. As a rule, the pupillary margin is torn but little, and the gaping can only be discovered by careful examination. Such lacerations are the most frequent cause of dilatation of the pupil, occurring after contusions, as they cause weakening or paralysis of the sphincter, due to laceration of its fibres. They frequently accompany simple cataract extraction (without iridectomy), and are caused by tearing of the iris in the efforts to remove the cataractous lens through an unyielding pupil. The iris and ciliary muscle may also be paralyzed by contusion, so that accommodation is affected.

Iridodialysis. Separation of the iris from the ciliary body is usually of considerable extent, involving even as much as one-half of the ciliary margin; it is usually single, but sometimes multiple. In the former we find two pupils, in the latter several. (Plate XIII., Figs. 13, 14.) The portion of the natural pupil toward the dialysis is flattened. In the iridodialysis we may see the edge of the lens, the zonule of Zinn, and the edges of the ciliary processes. As a rule, the sight is but little affected, although if the dialysis be so great that the torn portion of the iris lies in the visual line, central vision may be affected. Monocular diplopia may occur, on account of images being formed through the several openings upon the retina.

Iridirœmia Traumatica. If the iridodialysis be of such extent that the iris becomes torn in its full extent from its ciliary attachment, it may fall down in the bottom of the anterior chamber, and later shrink to an inconspicuous gray mass. If rupture of the sclera in the

ciliary region be produced at the same time, the iris may extrude or be expelled from the eye.

Inversion of the iris consists in its being pushed and turned back so as to lie upon the surface of the ciliary body, and it looks as if it were absent; partial dislocation is more frequently observed, and here the iris seems to be wanting, a coloboma appearing to exist. Total inversion is very rare.

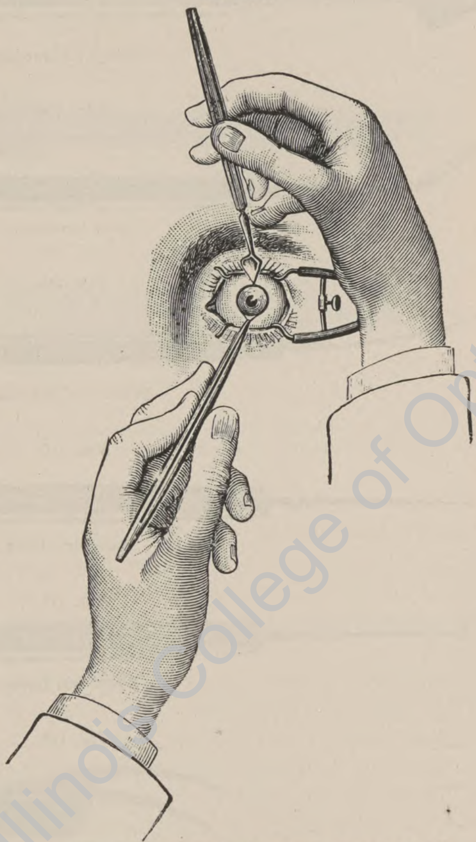
Hyphæmia or *hemorrhage into the anterior chamber* is due to and generally accompanies wounds of the iris. (Plate XIII., Fig. 16.) It is particularly marked in contusions of the eyeball and in operations on irides that have been previously inflamed. The blood sinks to the bottom of the anterior chamber, and disappears by resorption within a few days, when we may determine the extent of the injury. It is sometimes impossible to find a solution of continuity of the iris structure. The subjective symptoms of hyphæmia depend upon its extent. If excessive, so that the tension is raised, considerable pain is complained of, and glaucoma may follow.

Causes of Traumatic Changes in the Iris. These are most frequently non-penetrating blows upon the eye in which two factors cause the injury: 1. The flattening of the cornea from the contusion, by which its circumference and also the insertion of the iris become larger. If this enlargement takes place suddenly, the iris does not adapt itself, and tears away in places from its insertion, so that iridodialysis is produced. 2. The cornea being flattened, pushes the aqueous backward against the posterior wall of the anterior chamber, which in the area of the pupil is formed by the lens, and in the rest of its extent by the iris. The latter, when pushed backward, finds its support in the lens, except in the marginal portion of the iris, where the posterior chamber is deepest; therefore, the periphery forms the most yielding spot, and is the first to give way before pressure. This bulges the iris back as far as the zonula, or even into the vitreous. Thus, a blow upon the eye may produce: (*a*) marked stretching of the vitreous in a radial direction; (*b*) dilatation of the pupil; (*c*) in extreme cases, rupture of the zonula. The first affection may cause iridodialysis; the second, radiating lacerations of the sphincter, and, consequently, paralysis of the pupil; the third affection subluxation or luxation of the lens, iridonesis, or tremulous iris. If the edge of the iris slip back over the lens, it may produce inversion of the iris, and the lens may be luxated into the anterior chamber. Lacerations of the pupillary edge may be produced during the simple operation for cataract—*i. e.*, without iridectomy where the sphincter pupillæ is rigid and does not permit of easy passage. Iridodialysis may also be produced in operations upon the iris; if the eye makes a violent movement or the iris be roughly grasped with the forceps, the iris has been known to have been entirely torn out during such an operation. In iridectomy done for occlusion of the pupil, the iris may be torn loose at its periphery if the adhesion at the pupillary area does not give way; hence the iris ought always first to be

released from the pupillary membrane before it is drawn out of the wound. Iridodialysis may also be caused by tumors of the ciliary body pushing the iris away from its insertion.

Treatment. Treatment of the above-described injuries to the iris (except penetrating wounds of the eye) depends largely upon the extent of the bleeding (hyphæmia) within the eye. If trivial, the injured eye may be bandaged for a few days and the patient kept in bed to ensure absolute quiet, so that further bleeding does not take place and the deleterious effect of the contusion may not be assisted by further detachment of the retina occasioned by ordinary movements of the body. If the hyphæmia be excessive, causing tension and pain, paracentesis of the anterior chamber may be necessary. Hot compresses applied at intervals, as in the case of iritis, assist in absorption of the exuded blood. If an iridodialysis can be made out, atropine should be instilled, so that the contracting sphincter does not draw the iris farther away from its attachment. It is contraindicated in radiating lacerations, as a mydriatic would make the wound gape more. Iritis does not usually follow non-penetrating injuries. The internal administration of alkaline purges and one or two pilocarpine (0.005 to 0.01 gm.) sweats during the first forty-eight hours seem materially to assist resorption of blood in the severe cases; iodide of potassium may be given later.

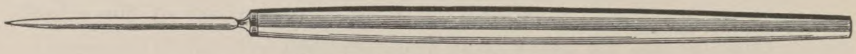
Fig. 170.



Position of operator's hands in first stage of iridectomy. (Czermak.)

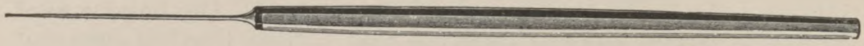
Operations upon the Iris. Formerly a number of operations were done upon the iris, but in modern practice only three forms are resorted to: 1. The removal of a segment of the iris for enlargement of the pupil. 2. Removal of a section of the iris and iritic membrane where the pupil has been closed by inflammation. 3. Incision into the iris or iritic membrane, in order to make a permanent opening or pupil. These operations involve incisions in the cornea,

FIG. 171.



Von Graefe linear knife (side).

FIG. 172.

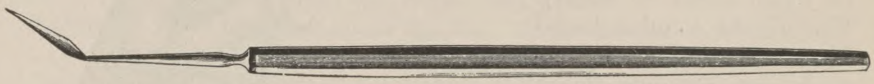


Von Graefe linear knife (back).

FIG. 173.

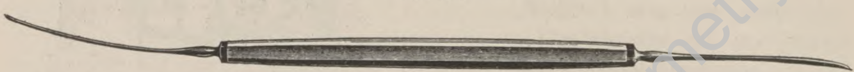
Straight keratome.

FIG. 174.



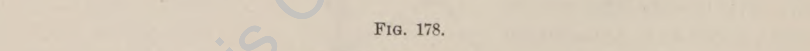
Bent keratome.

FIG. 175.



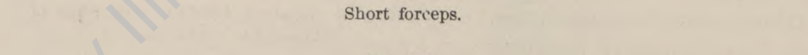
Probe and spatula.

FIG. 176.



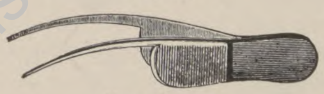
Sharp iris hook.

FIG. 177.



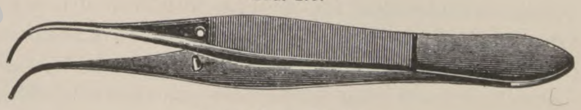
Blunt iris hook.

FIG. 178.



Short forceps.

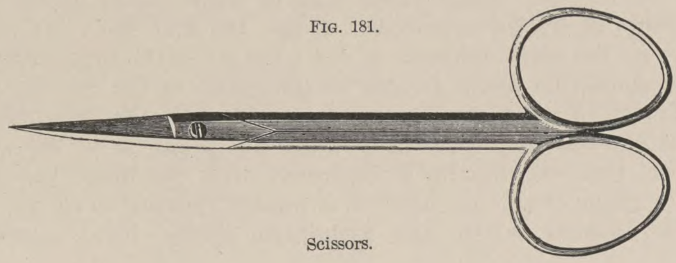
FIG. 179.



Long, bent forceps.

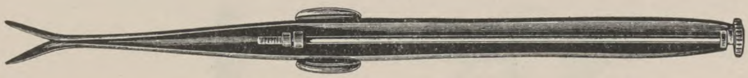
FIG. 180.

FIG. 181.



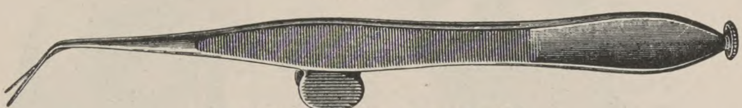
Scissors.

FIG. 182.



De Wecker's iridotomy scissors (front).

FIG. 183.



De Wecker's iridotomy scissors (side).

Instruments used in operations on the iris (slightly reduced). Other instruments required are a spring speculum or Desmarres' retractor, to keep the lids apart, and a fixation forceps to hold the eyeball.

and penetrating wounds of the eyeball, which, if aseptic, heal without inflammation; but if septic are attended by all the dangers of septic penetrating wounds of the eye. As sepsis occurs in the hands of careful operators in but 0.5 per cent. of cases involving opening the eyeball, the danger is comparatively small.

Operations for Enlargement of the Pupil. IRIDECTOMY is the removal of a segment of the iris for the purpose of enlarging the pupil. The method of performing the operation is as follows: The patient's head, face, and eyes are prepared for the operation as if for cataract extraction. In eyes with considerable tension, particularly in inflammatory glaucoma, general anæsthesia is advisable, as local anæsthetics in these cases are not sufficiently penetrating to affect the iris; in other cases local anæsthesia is obtained by 1 per cent. holocaine solution or 10 per cent. cocaine solution, dropped several times upon the cornea. The instruments necessary are a speculum or Desmarres' retractor, to keep the eyelids apart, a fixation forceps to hold the eye, and other special instruments mentioned in the following description of the operation. (Figs. 171–183.)

At least one skilled assistant is needed. The patient being prepared, the operator steadies the eye by seizing the conjunctiva and episcleral tissue with fixation forceps, either above and behind the limbus and the proposed incision, or at the inferior circumference below the limbus. The incision may be made either by the Graefe lance knife or by the keratome. (Figs. 184 and 185.) If with the former, in the same manner as for cataract extraction, except that the cut should be made as near to the plane of the iris as possible, and, hence, mostly in scleral tissue. If with the keratome, the knife is entered perpendicularly until the point is in the anterior chamber; then the handle is depressed until the blade lies parallel with the plane of the iris, when it is pushed forward until the wound is of the desired length, and withdrawn slowly, being sawed from

FIG. 184.

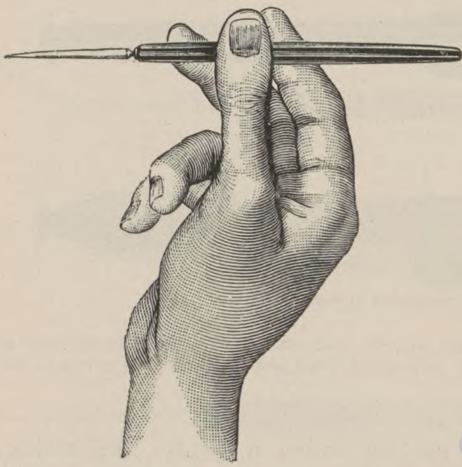


FIG. 185.

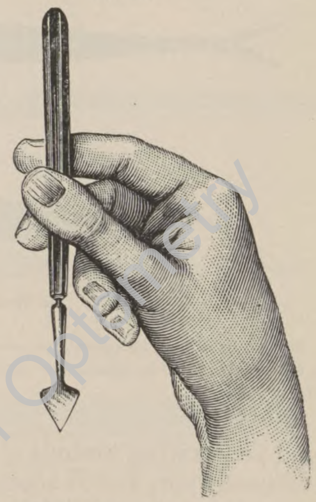


FIG. 184.—Method of holding the straight Graefe knife for upward incision. (CZERMAK.)

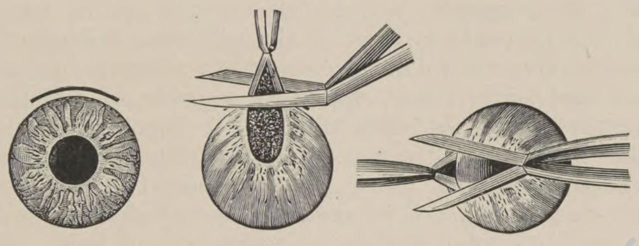
FIG. 185.—Method of holding the bent keratome for incision of the upper corneal margin. (CZERMAK.)

side to side, so that the ends of the linear incision on the inner surface of the cornea are made slightly larger, and so that the aqueous comes out slowly. During withdrawal the instrument is pressed against the posterior surface of the cornea, so as not to injure the iris or lens, which push forward as the aqueous flows off. After completing the incision, the closed branches of the iris forceps are introduced into the anterior chamber and pushed onto the border of the pupil; the branches are allowed to separate, and the fold of the iris is grasped gently, pressure and traction being made, which draw the iris out of the wound. At the moment when it is stretched, it is cut off close to the wound with the curved scissors or with the scissors forceps, either by one cut from above or incisions made to either side. The iris may be withdrawn by the iris hook, if but a

small section is desired, the forceps being generally better in glaucoma operations, and the iris hook in cataract or optical iridectomy. The ends of the wound are freed from the iris, and the spatula being introduced into the wound puts back into the anterior chamber any iris tissue and clears away the blood and débris. At the completion of the operation the pupil and the coloboma have the shape of a keyhole.

The eye operated upon, and usually its fellow should be lightly bandaged, the dressings being a semilunar piece of adhesive plaster on the upper eyelid, to act as a splint, sterilized vaseline to lashes to prevent them from gumming together, small patch of lintine or cheese-cloth to protect the eye from the dry absorbent cotton, which is placed over the eye and orbit, and over all a wire mask made to fit the face, or a light roller-bandage. The first dressing need not be made for forty-eight hours, when the eyelids are washed with boric acid solution and a light roller-bandage applied to the eye that has been operated upon, the other being protected by a reading

FIG. 186.



Iridectomy. *a*. Peripheral incision, as in glaucoma. *b*. Wide iridectomy. *c*. Narrow or optical iridectomy. (CZERMAK.)

shade. Atropine solution is usually dropped in at this and the subsequent two or three dressings, which are made at twenty-four-hour intervals, to dilate the pupil against the possible occurrence of traumatic iritis. (As there is excellent filtration and hypertension is not possible for a week or more after the corneal incision, or until the wound has fully healed, atropine is not contraindicated, even after glaucoma operations.) Dark glasses should be worn for several weeks, and at the third or fourth dressing absorbent cotton may be placed over the eye operated upon, and the dark glasses placed over this. Incision for glaucoma is made in scleral tissue as close to the root of the iris as possible; it should embrace at least one-fifth of the limbus. Incision for optical iridectomy usually is made in corneal tissue. The section for glaucoma and cataract extraction usually is made upward; that for optical purposes downward and inward, or in the region of least opacity. (Plate XIII., Figs. 5 6, 7.)

Indications for Iridectomy. 1. The most important indication for iridectomy is increase of tension in primary glaucoma and in sec-

ondary glaucoma resulting from exclusio pupillæ, ectasia of the cornea, or sclerochoroiditis or iridochoroiditis. The earlier the operation is performed, the better the success. Yet in some cases, in order to relieve pain and further degeneration, in ectasis of the eyeball in which perception of light has been abolished, the operation may be done. In iridectomy made after increase of tension the section should be made long, situated as far back as possible in the sclera, and the coloboma should be broad, and extend to the ciliary margin of the iris, for the reason that the results of iridectomy for the reduction of tension are achieved through the establishment of filtration through the scar tissue as well as by the removal of a portion of the actual contents of the eye in the portion of iris that is excised. The coloboma is made upward, so as to be covered partially by the upper lid, and the confusion due to dazzling thus lessened.

2. Operation on account of *optical obstructions*. The formation of an artificial pupil by iridectomy may be done in cases where opacities of the refractive media occupy the area of the pupil; among these are opacities of the cornea, membrane in the pupil, opacities of the lens that are non-progressive, in shrunken cataracts, which do not extend far toward the periphery, and in subluxation of the lens, where the pupil may be made in front of the part that contains no lens. To obtain results, the following conditions must be present: (*a*) the retina and optic nerve should be capable of functionating; (*b*) the opacity should be stationary; (*c*) the opacity should be so dense that it prevents the formation of distinct images upon the retina. These conditions are to be ascertained by proper observation and examination. Contraindications to iridectomy for optical purposes are: (*a′*) total absence of perception of light; (*b′*) strabismus of the eye affected by the opacity when the eye is not put in alignment; (*c′*) flattening of the cornea, which is an evidence of iridocyclitis and membranous exudates upon the iris; (*d′*) incarceration of the iris in a cicatrix, where the iris is primarily attached to the posterior surface of the cornea. Iridectomy for optical coloboma is made preferably downward and inward, if the media are everywhere equally transparent, as in the case of central cicatrix of the cornea, pupillary membrane, or perinuclear cataract, as the visual axis cuts the cornea a little to the inner side of the apex. In other cases the coloboma should be made at the place where the media are most transparent. Where only the most exterior marginal portion of the cornea remains transparent, the iridectomy may be made quite peripheral, involving the root of the iris; but, as a rule, it is made as narrow as possible, and only the sphincterial area of the iris is excised. (Plate XIII., Fig. 7.)

3. In ectatic cicatrix of the cornea, iridectomy is made to cause flattening.

4. In recurrent iritis, iridectomy made during an interval in which there is no inflammation sometimes prevents recurrences.

5. In fistula of the cornea, in cases where some trace of the anterior

chamber has been restored, iridectomy allows of the formation of a firm cicatrix by temporarily reducing the tension.

6. In the case of foreign bodies and small tumors of the iris, which sometimes can only be removed by removing the part of the iris in which they occur.

7. Iridectomy is done by some operators under nearly all circumstances in operating for cataract as they do the "combined" operation; in other cases, where delivery of the lens is impossible or difficult, on account of rigidity of the sphincter or a very large nucleus, it is necessary to excise a portion of the iris. In these cases only a small sector should be removed, and this should involve only the sphincterial area. These operations are made upward, as preliminary to the operation for cataract. Many operators perform preliminary iridectomy two or three weeks before removal of uncomplicated cataracts. Iridectomy should always be done in dealing with cataracts complicated with synechia, increase of tension, etc.

8. After excising the iris, the opacity has been found to mature more rapidly, especially if the lens fibres be massaged through the cornea.

Operations for Occlusion of the Pupil. 1. Removal of a Portion of the Iris and Iritic Membrane. Iritoectomy. (Fig. 187.) Occluded pupils may be opened after the method of de Wecker by an incision through the cornea, after which the forceps scissors are passed into the wound, one blade being forced through the iris tissue and the other remaining in the anterior chamber; a snip is then made and repeated twice, so that a triangular section of the iris is cut out and removed with the forceps or iris hook.

Fig. 187.

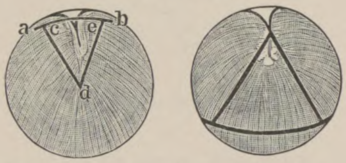


Fig. 188

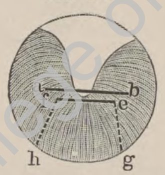


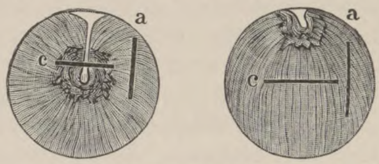
Fig. 187.—Iritoectomy. *a–b.* Corneal incision. *a–d, b–d.* Iris incisions. (de Wecker.)
Fig. 188.—Iridodialysis. *a–b.* Corneal incision. *f–e, f–h, e–g.* Iris incisions. (de Wecker.)

Iridodialysis (Fig. 188) as an operative procedure is done by a horizontal incision through the cornea with the Graefe knife or the keratome. The iris being divided horizontally at the same time, the forceps scissors are then introduced, cutting the iris on either side of the wound to the sclerocorneal margin; the flap thus cut is then torn out by grasping it with the iris forceps or sharp hook. This is a more dangerous operation than the preceding.

2. Incision of the Iris and Iritic Membrane. Iridotomy consists in simply dividing the iris without excising a piece. The incision into the iris is adapted only for those cases in which no lens is present; for instance, as an after-operation for cataract cases in which

the pupil has become closed by subsequent iridocyclitis. The operation is done by passing the Graefe knife perpendicularly through the cornea, rotating it 90 degrees, making a horizontal incision through the iris. (Fig. 189.) The edges of the opening retract, leaving the new pupil more or less circular.

FIG. 189.

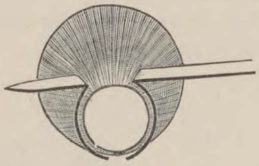


Iridotomy. *a*. Corneal incision. *c*. Iris incision. (DE WECKER.)

3. STAPHYLOTOMY for opening a closed pupil, caused by incarceration of the iris in a corneal cicatrix, is done by a sweep of the Graefe knife through the anterior chamber. (Fig. 190.)

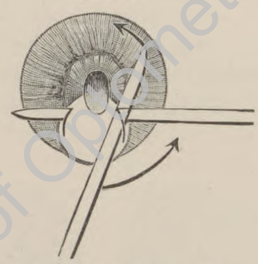
SPHINCTEROLYSIS ANTERIOR (Fig. 191) is done by transfixing the ectasia by one Graefe knife, another being passed into the eye and swept around, dividing the iris, and producing a more or less irregular pupil.

FIG. 190.



Staphylotomy. (ABADIE.)

FIG. 191.

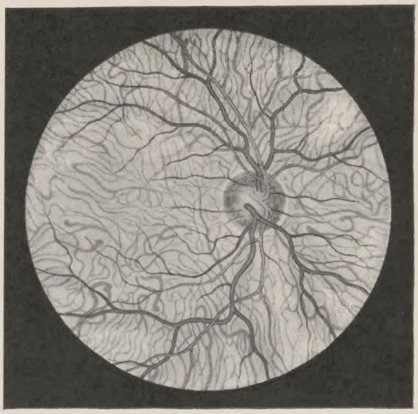


Sphincterolysis anterior. (SCHULEK.)

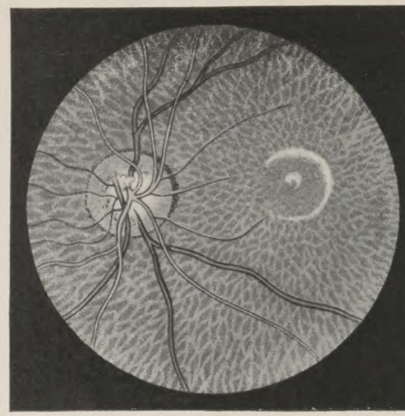
Formerly other operations were practised, such as *iridesis* which consisted in moving the pupil to one side by allowing the iris to become incarcerated in a corneal incision, and *corelysis* or division of posterior synechiæ; but, as their results are dangerous, or the operations are unnecessary, they have fallen into disrepute. As operations done for opening occluded pupils are, as a rule, made through pathological products or diseased irides, their results are often only temporary, the artificial openings closing later from recurrence of inflammation. Operative procedures may have to be repeated, and often are made in vain.

The after-treatment of the foregoing operations is similar to that following iridectomy.

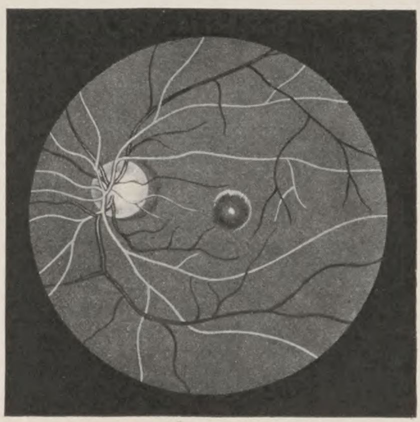
PLATE XIV.



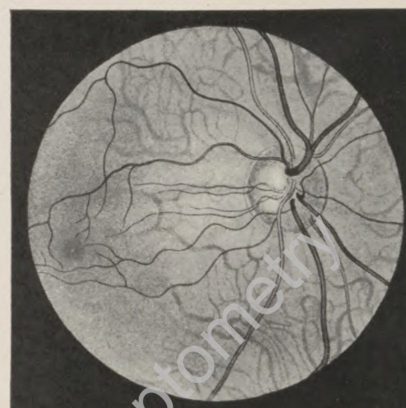
A



B

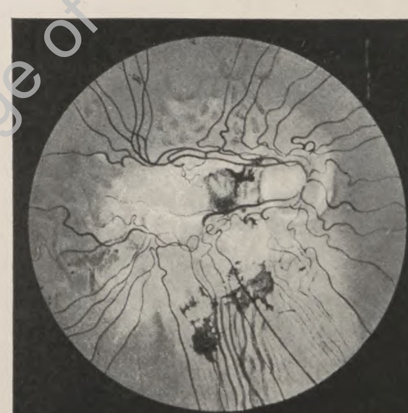


C



D

E



F

Anomalies of the Choroid.

A. The Albinotic Fundus; (Type I., see Plate XII., E).
B. The Tessellated Fundus; (Type II., see Plate XII., F).
C. The Negroid Fundus; (Type III., see Plate XII., G).
D. The Yellow Fundus; (Fundus Flavus, Oeller).
E. Sarcoma Choroideæ; V=-6/2×; (Hemianopic Field, see Fig. 197).
F. Coloboma Choroideæ Centralis; (Oeller) (Central Scotoma).

DISEASES OF THE CHOROID. CONGENITAL ANOMALIES.

Variations in the Color of the Fundus. Aside from the three types of fundus due to variations in the pigment cells, there may be congenital absence of pigment in albinism. Such a fundus is shown in Plate XIV., A. On account of the lessened contrast between the optic nerve and other parts of the fundus, the nerve head appears of a dark reddish-gray color; not only the retinal, but all the large vessels of the choroid are readily seen. Such persons have yellowish-white flaxen hair, white eyebrows and lashes, and their eyes are photophobic, visual acuity is reduced, and nystagmus is constantly present, and, as a rule, there are strabismus and myopia. Such a case affords a decided contrast from that of the negroid type. (Plate XIV., C.)

Treatment. Correction of the refraction and the use of shaded glasses, to protect the eye from brilliant light. Even with such assistance, such patients are usually unable to pursue ordinary vocations.

According to the disposition of the choroidal pigment, the fundus takes on more or less color, as in the several distinct types noted in Plate XIV., A, B, C, D. The result of inflammations is frequently an increase in and deposit of the choroidal pigment in spots, and from this, great changes in the appearance of the eyes are observed.

Coloboma of the Choroid. This anomaly (Plate XV., F) is due to a circumscribed defect in the choroid resulting from failure of closing of the fœtal cleft, usually in the retina, generally situated below the optic disk. If unaccompanied by coloboma of the ciliary body and of the iris, it may be of an oval shape; if with that of the ciliary region and of the iris, it is that of a triangle with the apex directed toward the papilla. Such eyes have a large sectorial defect in the visual field in the upper portion, and the central visual acuity is usually less than normal. (Fig. 193.) Coloboma of the choroid is associated usually with other anomalies of development, commonly with microphthalmos and coloboma of the iris. Nearly all such eyes are hyperopic.

A somewhat rare form of coloboma is a defect in the choroid at the region of the macula lutea (Plate XIV., F), and with this there is generally defect of the retina at this point, and consequent central scotoma. All such cases that I have seen have been associated with optic nerve atrophy and evidence of prenatal choroiditis. In both forms of colobomata the edges are sharply defined and commonly bordered by pigment. The bloodvessels pursue a very irregular course, the retinal vessels usually avoiding the coloboma in running along its edges.

Treatment. There is, of course, no treatment for these defects, but errors of refraction may be neutralized to advantage.

Inflammatory Diseases. *Hyperæmia of the choroid* is not in itself recognizable as a lesion or a symptom; as it is the first stage of inflammation, it undoubtedly is an accompaniment of most choroidal affections.

Choroiditis may be exudative or suppurative.

Choroiditis Exudativa. Acute exudative choroiditis is characterized by isolated foci of inflammation scattered over the fundus, appearing upon ophthalmoscopic examination as indistinctly outlined yellowish spots lying beneath the retinal vessels in the red fundus. (Plate XV., A.) These are due to infiltration of the choroidal substance with exudation, hiding the choroidal vessels; the overlying retina usually is involved, and, being clouded, covers the choroidal mass with a faint grayish veil. Isolated hemorrhages may appear in the choroidal stroma or under the retina. The exudates may pass not only into the retina, but also into the vitreous; thus opacities of the vitreous are almost always constant accompaniments of choroiditis, and the disease is really a compound affection.

Deposits upon the posterior surface (descemitis) of the cornea in a large number of cases of apparently simple exudative choroiditis show that the disease is a true uveitis, and is not limited to the choroid proper.

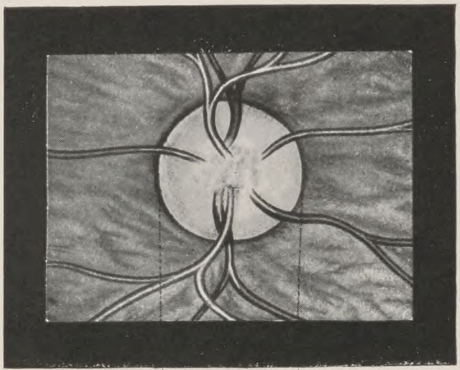
Symptoms. Subjectively, the patient complains of loss of visual acuity and of floating spots, which are due to complete or partial scotomata from implication of the retina and vitreous, and of flashes of light, and photophobia, due to irritation of the retina. As there are no sensory nerves in the choroid, there is no pain in uncomplicated cases.

The limitation of vision both as regards visual acuity and the visual field is not a prominent symptom; indeed, severe inflammation may be present without these visual factors being appreciably affected, until the chronic stage or that of atrophy, where degenerative spots and increase of pigment appears, and the retina and optic nerve become involved; then the visual acuity and field suffer. (Fig. 194.)

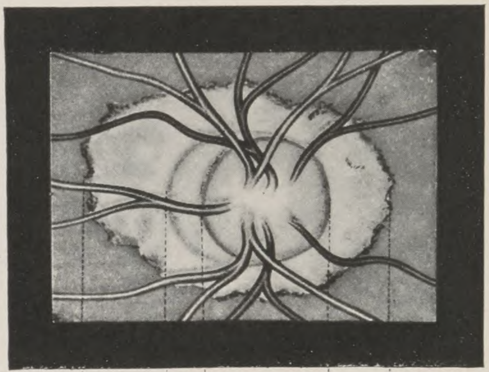
Course. While the course of choroiditis is essentially chronic, it may be subdivided into an acute stage, marked by inflammation and exudation, which last for several weeks or months, and the chronic stage, or that of atrophy, which lasts for months or years.

The chronic stage, or that of atrophy, presents a radically different picture: As the exudates become resorbed, the spots become more prominent and lighter in color, and gradually a white spot is formed as the choroidal stroma atrophies, which is due to the white sclera showing through. In some cases the remains of the vessels and the pigment may be recognized in the white cicatrix. The pigment generally proliferates around the edges of these scars, so that the choroiditic plaques appear lined with black, or covered with black spots. (Plate XV., B.) The visual acuity and the field suffer greatly.

PLATE XVI.



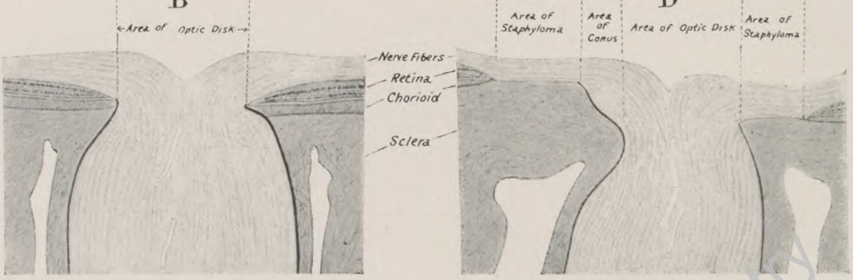
A



C

B

D



NORMAL OPTIC NERVE ENTRANCE.

A. Ophthalmoscopic view.

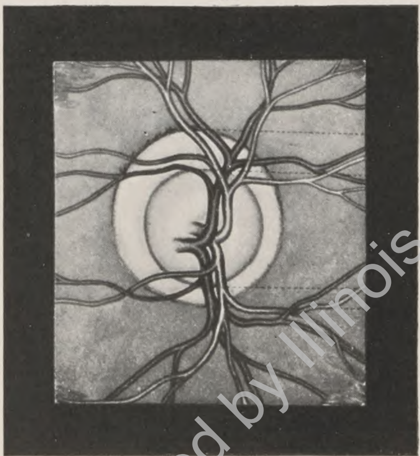
B. Diagrammatic section.

POSTERIOR CHORIOIDAL STAPHYLOMA WITH SICKLE-SHAPED CONUS.

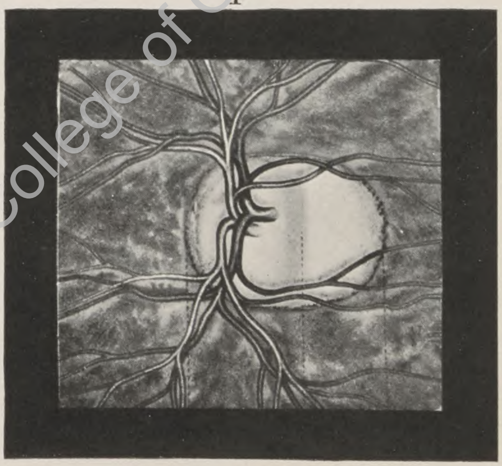
C. Ophthalmoscopic view.

D. Diagrammatic section, showing retraction of the chorioid.

E



F



RING CONUS.

LARGE SEMILUNAR CONUS.

The Optic Nerve Entrance in its Relation to the Choroid.

Etiology. Exudative choroiditis is a frequent disease, and is seen at all ages. Its causes are local irritations and disturbances of the nutrition of the eye, due to eyestrain, errors of refraction, irritation due to exposure to bright light, and to general disturbances of nutrition, such as anæmia and chlorosis. Thus it is accompanied by general diseases, particularly syphilis and scrofula.

Myopia of high degree is accompanied also by changes in the choroid less of inflammatory character than of atrophic; these are caused by stretching and tearing of the choroid at the optic nerve entrance. (Plate XVI.)

In moderate degrees of myopia, from —5 D. to —6 D., as a rule, only one side of the optic nerve entrance is affected, forming a conus (Plate XVI., F); in severe degrees the posterior section of the eyeball bulging backward, the choroid retracting considerably with the sclera, forms not only conus, but also posterior staphyloma. (Plate XVI., C, D, E, and Plate XV., E.) Such cases are accompanied by localized choroiditis at the edges of the coloboma, with increase in the choroidal pigment ring; changes likewise occur at the macula, and the pigment granules of the retina are more or less absorbed, allowing the intravascular pigment of the choroidal stroma to show. An eye affected by choroiditis of any degree of severity always loses more or less of its function, as may be determined by careful examination of the visual acuity and of the field.

Treatment. The treatment of choroiditis is that of its cause. Eyestrain should be relieved by proper lenses and unhygienic habits corrected; particularly is this the case in myopia, where full correction of the error should be given for distance, and the ciliary muscle favored by weaker lenses for the near. In most cases the glare of light should be mitigated by the wearing of smoked glasses. In acute cases atropine solution of sufficient strength and often enough to suspend accommodation should be instilled into the eye. Hot compresses favor resorption of exudates, and may be used three or four times a day for half an hour at a time. Injection of salt solution under the conjunctiva is used for the same reason. In acute choroiditis, extraction of blood by the natural or artificial leech, applied on the mastoid process over the emissary vein of Santorini, which comes from the cavernous sinus, into which the ophthalmic veins pour their contents, is useful. In the chronic stage, massage of the eye with the finger-tips once or twice a day for five minutes at a time stimulates metabolism. The general treatment should be directed against the exciting cause; disturbance of nutrition from anæmia should be met by exhibition of iron and arsenic, proper diet, and regimen. When the disease is accompanied by the rheumatic or uric acid diathesis, administration of sodium salicylate (0.50 to 0.1 gm. three times daily), or oil of wintergreen (0.30 to 0.50 c.c.) is indicated. Cystogen or urotropin (0.30 gm. three times daily) rapidly relieves the system of uric acid. When the patient is scrofulous, alteratives, tonics—*i. e.*, mercury, gold, arsenic, and iodide of iron

with cod-liver oil—should be exhibited. If the local lesion be a symptom of syphilis in the acute stage, mercurial inunctions will often produce a quick effect; the salts of mercury, gold, and arsenic seem to be specifics for this affection; iodide of potassium is particularly serviceable in resorbing exudates in the retina and vitreous. Diaphoresis by vapor baths and pilocarpine are of use when properly carried out.

FIG. 192.

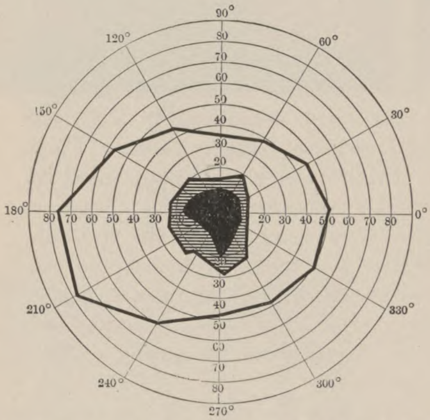


FIG. 193.

FIG. 192.—Central absolute and relative scotoma due to hemorrhage in syphilitic chorioretinitis (Plate XIV., E); also typical of choroiditis centralis senilis (Plate XIV., C). (Visus = objects in periphery of field; no central vision.)

FIG. 193.—Sectoral contraction due to choroidal coloboma, and enlarged blind spot from posterior staphyloma. (Visus = 6/XXIV.)

FIG. 194.

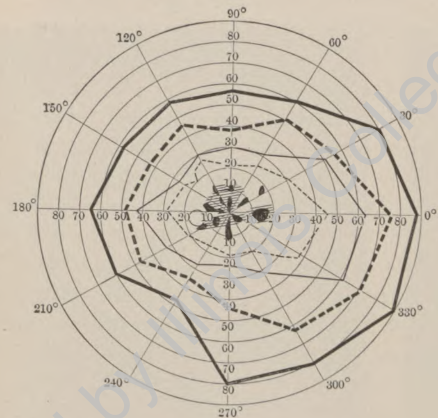


FIG. 195.

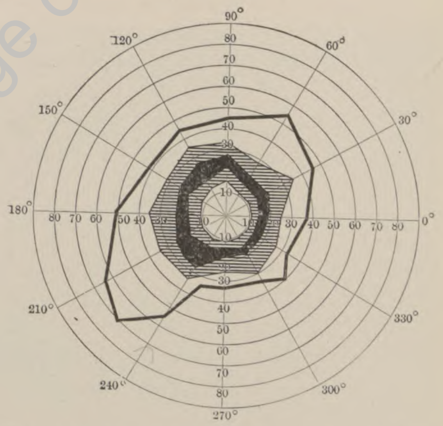


FIG. 194.—Paracentral and pericentral scotomata in choroiditis disseminata chronica (Plate XIV., B). (Visus = 6/LX.)

FIG. 195.—Absolute and relative ring scotomata in chorioretinitis diffusa syphilitica. (Plate XIV., D). (Visus = 6/LX.)

FIG. 196.

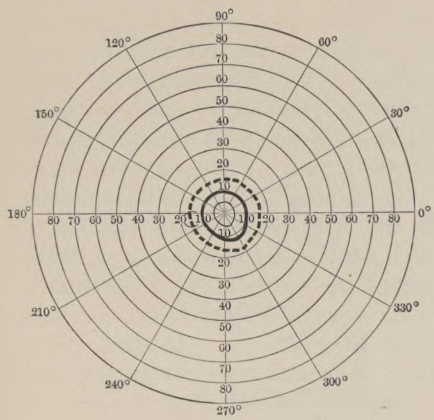


FIG. 197.

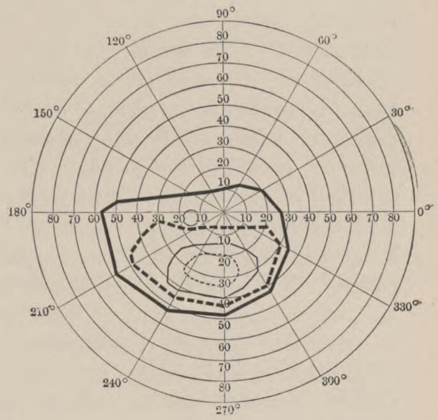


FIG. 196.—Great contraction in chorioretinitis pigmentosa. (Visus = 6/XII.)

FIG. 197.—Sectoral contraction simulating vertical hemianopia in sarcoma of choroid in first stage. (Visus = 6/XXXVI.)

Varieties of Exudative Choroiditis. Aside from the distinction of recent and old choroiditis, certain well-defined forms are to be differentiated:

1. *Choroiditis centralis* is characterized by changes occurring in the region of the macula lutea, causing disturbance of central vision from central scotoma, with resultant diminished visual acuity. (Fig. 192.) The most common form is that occurring in old people, affecting both eyes about equally, and is referable to senile changes due to sclerosis of the central vessels. In young people sclerosis of the vessels is not seen. In some cases there is considerable deposit of pigment as well as atrophy of the choroid. (Plate XV., C.)

2. *Choroiditis Areolaris.* The first foci develops in the vicinity of the fovea, while subsequent ones make their appearance at constantly increasing distances from the latter. The most recent spots are entirely black, and afterward slowly enlarge, at the same time becoming decolorized in the centre, at last becoming almost entirely white. One or two isolated spots may be seen in other portions of the fundus. This is perhaps a variety of the disseminated form. Myopia of high degree is accompanied usually by changes in the fovea. The acute form of macular choroiditis is found most often in syphilis; this subsequently degenerates into atrophy, with increase of pigment deposit. Injuries, such as contusions, entry of foreign body into the vitreous, burning of the macula from direct exposure to the sun's rays, as in observing an eclipse and electric light flashes, are liable to develop macular disease. The macular region of the retina and of the choroid is a vulnerable spot, and is affected readily by local or general diseases. Injury to it has a most deleterious effect upon the visual act, causing central scotoma and great loss of visual acuity. (Figs. 192, 194, and 195.)

3. *Choroiditis disseminata* is characterized by numerous round or irregular spots scattered over the fundus, composed of isolated inflammatory foci which at first look whitish, being accompanied by disease of the overlying retina. (Plate XV., A.) Some of these spots progress to atrophy, with increase of the retinal and choroidal pigment, while fresh ones appear, so that ultimately the eyeground appears studded over with the plaques, and in old cases a large portion of the fundus looks whitish. In the beginning the optic nerve and retina usually are involved in the hyperæmia, and the nerve looks reddish; ultimately atrophy of both the nerve and retina takes place. (Plate XV., B.) When many of the spots become confluent, the whole background of the eye may appear mottled and present a peculiar picture, resembling choroiditis diffusa. In the acute stage the visual acuity and the visual field suffer but little, so that nearly normal vision exists, the patient complaining mostly of dazzling and flashing of light, and asthenopia. As this is an essentially chronic disease, it ultimately progresses to amblyopia. From the changes taking place in the retina and optic nerve, there are usually scotoma and contractions of the visual field, with reduction of the visual acuity. (Fig. 194.)

4. *Choroiditis anterior* is characterized by exudation at the periphery of the choroid, the fundus being here studded with roundish ink-black opacities. It is found in myopes of high degree and sometimes as an accompaniment of syphilis. In old people pigmentary changes are frequently found in the anterior portion of the choroid. It is sometimes a complication of retinitis pigmentosa.

5. *Choroiditis Diffusa.* In recent cases the retina and vitreous appear cloudy, and circumscribed exudates are present in the retina and choroid, occupying mainly the region of the macula. Later on, these appear as dirty light-gray irregular patches, and in the last stages the general cloudiness of the media disappears, being replaced by atrophy in the retina and choroid, into which migration of pigment takes place. (Plate XV., D.) This is a disease characteristic of syphilis, and has been described under the name of choroiditis syphilitica. The visual acuity and field suffer greatly, color vision particularly being affected. (Fig. 195.)

6. *Tubercular deposits* rarely occur in the choroid; they present the picture of yellowish-white plaques, over which the retina appears grayish and infiltrated. (Plate XVII., A.) These ultimately degenerate into atrophy, pursuing much the same course as the disseminated form.

7. *Changes in the choroid in myopia* consist (*a*) in retraction of the choroid and atrophy at the border of the optic disk. This first appears as a crescentic patch at the outer border of the papilla, being caused by the head of the nerve being pulled toward the temporal side. Later on, the scleral canal enclosing the nerve is so pulled and gets such a slant that upon ophthalmoscopic examination it comes into view through the transparent tissue of the papilla, ap-

pearing as a white crescent close to the temporal border (Plate XV., E, and Plate XVI., F); this is called conus. At this time a reflex may be seen in some cases at several disk diameters temporally from the nerve head, which is a characteristic symptom of progressive myopia, the curvilinear line of Weiss. As the myopia progresses the retraction extends around the nerve, forming a band of white tissue—ring conus. (Plate XVI., E.) If the retraction extends further, inflammatory changes, with consequent atrophy, take place, resulting in bulging back of the ball or posterior staphyloma. (Plate XVI., C.) (*b*) High myopia is likewise accompanied by inflammatory changes about the macula, such as have been described under the heading of Choroiditis Areolaris.

Complications. In mild cases of choroiditis, the retina and optic nerve may become hyperæmic. When the affection is due to eye-strain, the cause may quickly be relieved by correction of the refraction, and thus the retina and optic nerve acquire no pronounced defects; but if choroidal disease exists for any length of time, or is severe, incidental inflammation and subsequent atrophy of the optic nerve and retina occur, with subsequent diminution of vision. Thus it is that most cases of choroiditis are a chorioretinitis. (Plate XVII., C.) The choroid likewise is generally involved in cyclitis and iritis, which are accompanied by hyperæmia or ulimate degenerative changes in the choroid. Iridochoroiditis has thus been described as a special disease. The vitreous is generally involved in exudative choroiditis, and the results of choroidal inflammation and exudation are to be seen therein in opacities of the vitreous.

Choroiditis Suppurativa. Suppurative choroiditis may originate in the choroid, being evident at first by a local exudation containing numerous cells and pus germs. The inflammation extends to the retina and vitreous, and at this stage may be seen through the pupil as a yellowish mass in the fundus, or later as a yellowish reflex. (Plate XVII., B.) The inflammation becomes violent, implicating the ciliary body and iris, and, finally, all the structures of the eye, causing panophthalmitis.

Symptoms. There are but few mild cases, but in these or in the beginning of suppurative choroiditis, although the inflammation proper is confined to the uvea itself, the media become clouded, and fundus examination is limited to the perception of a yellowish glimmer. The vision is greatly diminished, there is pain, and, on account of the iritis, congestion is present. There may be slight rise of temperature. In the graver cases the inflammatory symptoms progress to severe implication of the ocular structures, violent pain is present, and sight is completely lost; hyperpyrexia likewise occurs.

Course. In the beginning there is hypertension, owing to exudation; the eyeball softens later, and finally atrophies. In severe cases the conjunctiva and eyelids become œdematous, and the conjunctiva often so chemosed that it projects between the lids, which can be with difficulty forced open. In violent cases the eyeball

protrudes and becomes immovable, from infiltration of Tenon's capsule and the orbital contents; pain becomes intolerable and tormenting photopsiæ are present; high fever exists and vomiting occurs; the inflammation may extend to the brain, and suppurative meningitis result. The symptoms increase until the purulent exudation in the eye makes an exit, a perforation of the sclera usually taking place in the anterior portion; the conjunctiva and sclera bulge forward; the purulent contents slowly extrude, the pain then ceases, and after six or eight weeks the eyeball shrinks to a small stump (phthisis bulbi).

Etiology. Choroiditis suppurativa is produced by infection of the choroid from pyogenic matter:

1. *Ectogenous infection* may arise in the choroid itself or from the outside: (*a*) Penetrating injuries and infected operative procedures involving opening of the eyeball are the most frequent causes. (*b*) The passage of suppuration from without inward in perforating ulcer of the cornea and prolapse of the iris. (*c*) Infection may pass in from corneal or sclerotic cicatrices, with incarcerations of the iris. The germs enter from a lesion in the epithelium covering the cicatrices or by sudden stretching or bursting open of the latter.

2. *Endogenous infection* occurs: (*a*) Through embolism from pyogenic substances passing from the general circulation into the vessels of the choroid, becoming there arrested, and developing metastatic choroiditis. This is a symptom of pyæmia. The most frequent form is seen in puerperal fever. It also occurs from infective suppurating glands, such as chancroidal buboes. (*b*) By extension of the inflammation from the meninges, particularly in the cerebrospinal form. The cases occur chiefly in children, and are distinguished by their comparatively mild course, so that in rare cases some small degree of sight may be obtained. (*c*) By extension of the inflammation from behind in phlegmonous inflammation of the orbit and thrombosis of the orbital veins.

Prognosis. The prognosis of suppurative choroiditis is very unfavorable. In practically all cases the sight is lost. In most cases the result is atrophy of the eyeball, and where the choroiditis is but one of the symptoms of pyæmia or meningitis the life of the patient is jeopardized.

Treatment. No medication can change the course of suppurative choroiditis. It is confined to ameliorating the patient's suffering. The pain may be combated with hot compresses and narcotics. If the case progresses to panophthalmitis, free incision of the sclera in its anterior portion diminishes the tension by allowing evacuation of the suppurating contents, and thus the pain and progress of the disease are cut short. When the eye becomes shrunken, it usually remains quiescent; but in some cases further degeneration, such as calcareous deposits, occurs, causing irritation of the ciliary nerves and sympathetic irritation in the other eye.

While an artificial eye might be worn over the resultant stump, still the irritation caused by the shell may give rise to secondary symptoms. Although it has been customary to do prosthesis over a shrunken stump, the danger from sympathetic irritation should be considered, and enucleation should be practised.

Enucleation in the height of panophthalmitis should not usually be done, except in cases where phlegmon of the orbit is likewise developed and where it is necessary to obtain thorough drainage. Suppurative meningitis has been reported as occurring after enucleation for panophthalmitis, but also several cases have been reported of fatal meningitis succeeding a panophthalmitis in which enucleation was not practised. The general treatment should be that adopted for septicæmia or pyæmia: quinine, alcohol, and strychnine are to be exhibited, and the excretory functions kept in normal condition.

Sequelæ. The result of severe plastic or purulent inflammation of the uvea is shrinking of the eyeball, due to absorption of its contents and their replacement by connective tissue. Two forms are observed:

FIG. 198.

Atrophy of the eyeball. The eyeball is smaller and of quadrangular shape, from pulling of the recti muscles, and grooved at their insertion; the cornea is irregular, the retina detached from the choroid, and much exudation in the remains of the vitreous behind the lens; the choroid remains attached at the posterior portion of the globe, being detached only as far as the ora serrata; between the choroid and the retina is a space filled with an albuminous fluid; the optic nerve is thinner and atrophic.

1. *Atrophy* (Fig. 198), where the shrinkage takes place slowly; the diminution is usually moderate, being caused by the contracting exudate. The tissues of the eye remain individually distinct. The shrinking of the exudate draws the intra-ocular contents together, causing repeated attacks of inflammation, and, at times, sympathetic irritation in the fellow eye. The cause of atrophy of the eyeball lies chiefly in plastic iridocyclitis. The atrophy goes on for months or years, and may result in phthisis bulbi.

2. *Phthisis Bulbi.* Here the shrinkage following perforating panophthalmitis is rapid, as a rule, the eye becoming very small, shrinking even to the size of a hazelnut. The ocular contents are extruded through the rupture or are rapidly absorbed; hence such eyes are seldom painful, and do not give rise to sympathetic irritation in themselves, but the original inflammation through which they passed, in many cases gives rise to sympathetic inflammation.

Essential Phthisis Bulbi (ophthalmomalacia). This is a rare affection, supposed to be due to a lesion of the sympathetic. The eyeball becomes soft, and in mild cases opacities of the media occur. The condition may last for several days or weeks, and finally disappear without leaving traces. In severe cases the tension becomes lowered and the eyeball permanently diminished in size.

Ossification of the choroid and ocular contents is found not infrequently in shrunken eyes of long standing; a thin shell of bone is found in the posterior portion, in which there is a hole for the optic nerve. Calcareous degeneration of the contents likewise may occur. The stump is often painful to touch, and may give rise to sympathetic irritation.

Treatment. Atrophic eyeballs containing foreign bodies or having undergone osseous or calcareous degeneration, and all irritable and painful stumps should be enucleated. In but few cases in which there are small non-irritable stumps, should artificial eyes be fitted without enucleation.

Neoplasmata of the Choroid. New-growths of the uveal tract are relatively uncommon, occurring in 0.0375 to 0.066 per cent. of eye cases. Of these, sarcoma is relatively common, and is seen most often in the choroid. It usually is pigmented, and its course is such that four distinct stages may be observed. If occurring

FIG. 199.

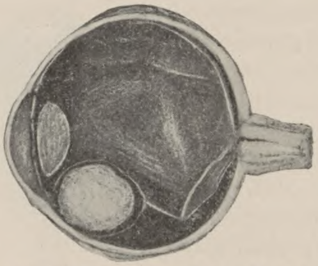


FIG. 200.

FIG. 199.—Sarcoma of choroid, first stage. (Photographed from specimen.)
FIG. 200.—Sarcoma of choroid; second stage; external aspect of enucleated eyeball.

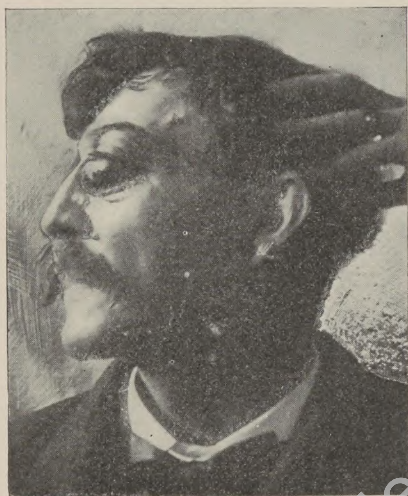
in the choroid, in the first stage the tumor is small (Fig. 199); and if occurring away from the macular region, does not at first affect the visual acuity, but gives rise to defect in the visual field from circumscribed detachment of the retina. (Figs. 192–197.) The diagnosis is usually readily made by the ophthalmoscope, although not always upon first examination, as it is to be differentiated from simple detachment of the retina. As the tumor grows the retina becomes more and more detached from the choroid, the space between it and the choroid containing a thickened, jelly-like mass infiltrated by connective-tissue elements and amœboid cells, but not with tumor tissue. The tumor itself is usually circumscribed, the choroid remaining in contact with the sclera in its full extent. Externally, the eye still appears normal. (Plate XIV., E.) On further growth, the eye becomes blind and ophthalmoscopic examination is impossible on account of the disturbance of the media.

In the second stage symptoms of increased tension set in, the eye presenting the appearance of inflammatory glaucoma, being inflamed, the cornea dull, the anterior chamber shallow, iris discolored, pupil

dilated and immobile, tension elevated. The lens later becomes clouded, and, besides the total blindness, the patient suffers pain. As these are the symptoms of inflammatory glaucoma, and, as many cases are not seen by the physician until this stage sets in, a correct diagnosis often is made with difficulty. If the eye be enucleated at this time, it may be found of an irregular shape from bulging of the ocular coats due to growth of the tumor. (Fig. 200.)

The third stage is that of perforation of the eyeball and extra-ocular growth of the tumor. Perforation occurs by infiltration of the sclera and replacement of its tissue by the neoplasm. The favored site is posteriorly, when the nodules of the tumor are invisible until after enucleation, but if anteriorly, dark and hard prominences may be seen developing in the region of the corneal limbus. As soon

FIG. 201.



Sarcoma of orbit including eyeball, originating in choroidal growth; third stage. (See Fig. 202.)

as the neoplasm has broken through the envelopes of the eyeball, the great tension and consequent pain usually ceases, and after this the tumor grows rapidly, filling the orbit with projecting cauliflower-like excrescences. (Figs. 201 and 202.) Ulceration of the superficial portion with hemorrhages and foul discharge then occurs. The neoplasm may extend directly into the neighboring parts and brain, the patient dying of septicæmia, from absorption of the necrotic products, from hemorrhage or from implication of the brain.

The fourth stage is that of metastasis and generalization in the internal organs, usually the liver. (Figs. 203 and 204.)

Metastasis begins during the second and third stages, and a diagnosis of internal disease cannot usually be made until the visceral

tumors are sufficiently large to be appreciated by palpation and percussion.

Sarcomata of the choroid consist of either round or spindle cells, or are a mixed form. They may be pigmented or non-pigmented, and usually contain many wide bloodvessels. As they develop from the external layers of the choroid, their microscopic appearance is the same as that of sarcomata of other vascular tissues. Sarcomata

FIG. 202.

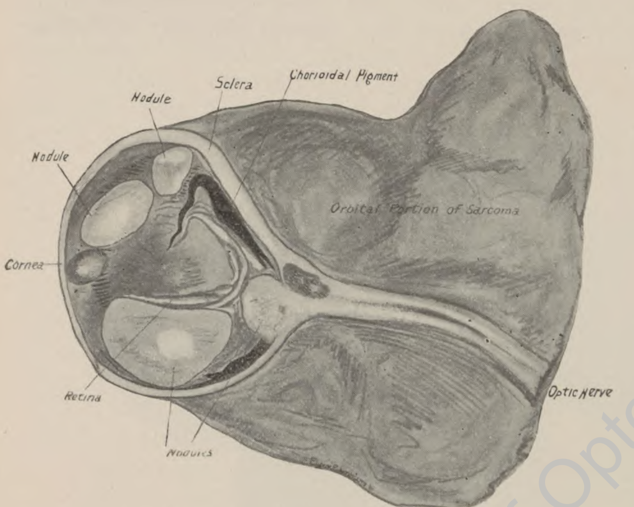


FIG. 203.

FIG. 202.—Sarcoma of orbit including eyeball, originating in choroidal growth; third stage. (From patient shown in Fig. 201.)
FIG. 203.—Metastatic or fourth stage of intra-ocular sarcoma. (Fig. 204.)

are the only forms of new growths that have been reported as occurring in the choroid.

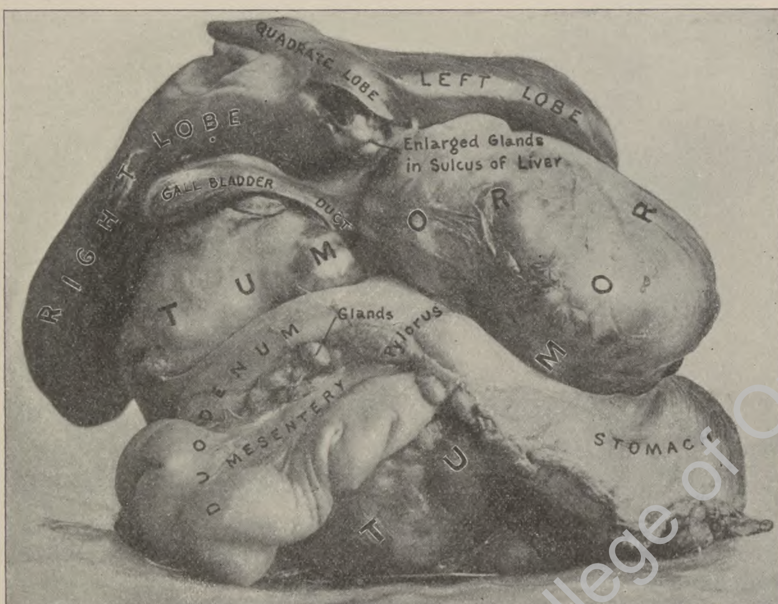
Duration and Prognosis. The first and second stages usually last three or four years. Patients die in the third stage from exhaustion or extension into the brain. The fourth stage is always fatal, and the two latter stages usually culminate in death in about a year.

Sarcoma affects the uveal tract and orbit in about equal proportions of males and females, the average age being forty-eight and one-half years. It is extremely rare in children, so that a malignant growth developing in an eyeball would, in all probability, be regarded as a

glioma in a child and a sarcoma in an adult. Recurrence takes place in about 8.86 per cent. In primary uveal sarcoma where the eyeball is removed early the prognosis is more favorable than when arising in the orbit, where recurrence takes place in 58.6 per cent. Sarcomata of the iris and ciliary body behave in respect to their course and ultimate outcome like those of the choroid.

Treatment. Early enucleation of the eyeball, in which the optic nerve is cut as far back as possible, is the treatment of the first stage. If the neoplasm has affected the orbit, complete exenteration—*i. e.*, removal of the entire contents of the orbit—together with the periosteum may be done in hopes to prolong life. Exposure of the

FIG. 204.



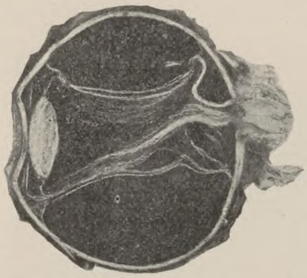
Abdominal contents in case of metastatic sarcoma originating in the eye, showing enormous secondary growth back of liver. (Same case as Fig. 203.)

denuded orbit to the action of the *x*-ray may then be resorted to as an additional security against recurrence. Recurrence and death, however, usually take place within two years, but a very small proportion of cases being cured by exenteration and *x*-ray exposure. The treatment of the fourth stage, where involvement of other organs has occurred, is simple palliation of the patient's suffering.

Injuries of the Choroid. *Penetrating wounds* of the posterior portion of the eyeball involve the choroid; they are likewise accompanied by injuries to the retina and vitreous. If clean, they heal by cicatricial tissue; if septic, inflammatory changes result, producing iridochoroiditis and panophthalmitis.

Rupture of the Choroid. This is produced by a contusion, usually by a blunt instrument; sometimes several, but generally only one laceration occurs. On account of extravasation of blood into the vitreous and under the retina, this injury is not usually recognized until some time after the accident, when healing has already taken place. (Plate VII., E.) Then, upon ophthalmoscopic examination, a white streak is observed over which the retinal vessels run without change in their course; the edges of the rent are observed to be colored by proliferation of the pigment; localized detachment or rupture of the retina is generally present. Constriction of the visual field and loss of visual acuity occur from atrophic changes in the retina and optic nerve.

FIG. 205.



Severe subchoroidal hemorrhage, with detachment of choroid and retina, following iridectomy for glaucoma. (Photographed from specimen.)

Treatment. Treatment of this condition is absolute rest in bed for a week or more, to reduce the liability of hemorrhage and retinal detachment, and instillation of atropine to quiet the action of the ciliary muscle and iris. Immediately after the accident saline cathartics may be given as derivatives, and iodide of potassium later, to aid in the absorption of clots or exudates.

Detachment of the choroid is due to subchoroidal hemorrhage, which gives symptoms of glaucoma (one of the so-called forms of hemorrhagic glaucoma). It is absolutely fatal to vision. This is one of the results of sudden relief of intra-ocular tension, and has been observed as an unfortunate accident after iridectomy, made in glaucoma, and also after cataract extraction.

Treatment. The eyeball usually has to be removed in order to stop the hemorrhage and great pain.

THE VITREOUS HUMOR.

Anatomy and Physiology. **Macroscopic Anatomy.** The vitreous (corpus vitreum) is a transparent, colorless, gelatinous mass filling the posterior cavity of the eye. It is surrounded posteriorly and laterally by the optic nerve and retina, and anteriorly by the ciliary body and capsule of the lens. On the anterior surface there is a depression, the fossa patellaris, in which rests the posterior surface of the lens. It is traversed from behind forward by the hyaloid canal, which is a lymph space beginning at the papilla and extending to the posterior pole of the lens. During fœtal life the hyaloid artery runs in this canal, and sometimes persists. The vitreous has no bloodvessels, and depends for its nutrition upon the surrounding

tissue, particularly the uvea; hence, affections of the inner membranes of the eye, the retina, and the choroid always implicate the vitreous.

Microscopic Anatomy. The vitreous is a transparent reticulum containing a clear liquid substance, with round or branched cells which are mostly found in the outer layer, and are supposed to be migrated white blood corpuscles. The external envelope is formed by a structureless layer, the hyaloid membrane.

The vitreous serves as a medium of support to the ocular tunics preserving the spherical shape of the eyeball, and as a clear medium permitting the passage of light and focusing of objects upon the retina. Its index of refraction is about the same as that of distilled water, which is 1.3340.

Diseases of the Vitreous.

Congenital Anomalies. *Persistent Hyaloid Artery.* The hyaloid artery passes from the central artery of the retina to the posterior surface of the lens, occupying the canal of Cloquet in the hyaloid canal during fœtal life, shrivels and disappears about the sixth month of gestation, but occasionally persists: (*a*) a filamentous strand attached to the disk or to the lens, the free end floating in the vitreous; (*b*) a strand passing across the vitreous; (*c*) irregular minute bodies upon the surface of the disk. Its vestigial remains are accountable for posterior congenital capsular cataract. There is, as a rule, no defect of vision, except it be accompanied by opacity of the lens or other congenital anomaly.

The walls of the canal of Cloquet are sometimes sufficiently opaque to be seen by the ophthalmoscope or to interfere with central visual acuity.

Inflammatory Diseases. *Hyalitis.* Inflammation of the vitreous is never primary, but accompanies and is the result of inflammation in the retina and the uveal tract. Vision is diminished from interference with the function of the retina, and also by disturbance of the media due to punctate spots in the vitreous, which the patient sees as floating specks. These are not to be confounded with muscæ volitantes, which are a normal phenomenon, being due to the amœboid cells in the vitreous, which are readily seen entopically by closing the eyelids and turning the face toward a strong light. Most patients annoyed by the latter have some error of refraction which should be corrected with suitable lenses. A fine, dust-like mist occurring in the course of choroiditis, particularly of the syphilitic variety, can be detected by the patient as floating specks and also by the ophthalmoscope. This is called hyalitis punctata. In another form of the disease they are seen as star-like or as minute light-colored spheres, asteroid hyalitis.

Opacities in the Vitreous. These are either fixed or movable, and are secondary to other affections of the retina and choroid. Large membranes may form as the result of hemorrhage or inflammation,

and are readily seen by the ophthalmoscope, impeding vision depending upon their location. (Fig. 206.) The ophthalmoscope offers a sure method of making the diagnosis if the media be clear. A convex glass of from 5 D. to 15 D. is used to focus the various depths of the fundus, and the patient directed to rotate the eye, by which the opacities may be brought into view, and their depth discovered by the strength of the focusing glass, and their paralactic

FIG. 206.

Striæ retinæ et membranæ vitreæ ex choroiditis exudativa.

movements. High degrees of myopia predispose to degenerations of the ocular contents, and usually are attended by membranes or opacities in the vitreous. The several diatheses and general diseases giving rise to diseases of the retina and choroid are likewise prone to develop vitreal opacities.

Treatment. The treatment of hyalitis and opacities in the vitreous should be that of the cause, if such can be ascertained. Alteratives, such as mercury and potassium iodide are sometimes useful. Irregularities of the menstrual function, disorders of the liver, etc., should be treated; diaphoresis with pilocarpine (0.01 gm. hypodermically) once a day, followed by a hot general bath, is sometimes useful. Errors of refraction and bad ocular or bodily habits should be corrected.

Hyalitis Suppurativa. This is an infective inflammation of the vitreous caused by entrance of pyogenic micro-organisms. It is an accompaniment of iridochoroiditis, and has been described under that heading.

Pseudoglioma. A circumscribed suppuration or plastic inflammation of the vitreous may occur in the periphery of the chamber near the ciliary region, being due to exudation from the ciliary body, and is accompanied by loss of vision and minus tension. On account of the yellowish reflex from the pupil, such cases have been mistaken for true glioma of the retina, but the evidences of a general uveitis, and the decreased tension should give the proper diagnosis.

Bloodvessel Formation in the Vitreous. After inflammation or hemorrhage into the vitreous, organization and development of bloodvessels may take place, forming a veil of freely communicating capillaries having seemingly no connection with the bloodvessels of the retina. These interfere with vision, depending upon the amount and their position.

Degenerations of the Vitreous. *Synchisis Corporis Vitrei. Fluidity of the Vitreous.* The vitreous being dependent upon the retina and choroid for its nutrition, during the progress and as the result of diseases of these membranes, and in high degrees of myopia, degeneration of the vitreous occurs, so that its framework is destroyed, losing its normal consistency and becoming a straw-like liquid. There are likewise diminished tension (hypotony) and frequently a tremulous iris (iridonesis), and occasionally a luxated lens. This condition is a most unfavorable factor for restoration of vision by cataract extraction. Treatment is of no avail.

Synchisis Scintillans. Cholesterin Crystals in the Vitreous. These are apparent to the patient by flashing sparks before the eyes and to the ophthalmoscope by numerous glistening crystals reflecting the light from the ophthalmoscope in the form of a shower of sparks. They are composed of minute crystals of cholesterin and tyrosin, and the ophthalmoscopic picture is very brilliant and interesting. As this happens in eyes that are more or less degenerated in other respects, the vision is reduced. The condition does not yield to treatment.

Fatty Degeneration of the Vitreous. In this condition there are muscæ volitantes, and the ophthalmoscope shows numerous white glistening spots evenly distributed through the vitreous. The vision is slightly reduced; but as this is an evidence of senile decay, there is no indication for special treatment.

Detachment of the Vitreous. The vitreous may shrink in volume from degenerative changes, and the retina thus losing its support becomes detached. It may arise from choroiditis, hemorrhage, extensive posterior staphyloma, and trauma. If the eye does not become inflamed, there is no occasion for treatment. If congestion, pain, or sympathetic disease set in, the eye may be enucleated.

Injuries of the Vitreous. *Loss of Vitreous.* Prolapse of the vitreous occurs as an unfortunate occurrence in penetrating wounds of the eyeball, especially in cataract extraction, and about one-fifth of the vitreous may be lost without materially affecting the function of vision, as the envelopes of the eye accommodate themselves somewhat to their diminished contents.

Treatment. If due to penetrating wounds of the sclera, the bead of vitreous may be cut off, the wound stitched, and the eye treated antiseptically; if occurring during cataract extraction, the toilet of the anterior chamber cannot be as rigidly made, and iris prolapses cannot always be replaced. The extruding vitreous should be snipped off, the eye closed, and disturbed as little as possible in the dressings. The extruded vitreous retracts somewhat, and, if it does not become infected, the wound heals, but union is delayed.

Hemorrhage into the Vitreous. This follows rupture of the vessels of the retina or choroid, most probably the latter, causing loss of vision depending upon the retinal and choroidal lesion and upon the amount of bleeding. Spontaneous hemorrhage may occur in young adults who have irregularities of circulation and gout. As a rule, these are not entirely absorbed, but leave opacities in the vitreous, damaging the vision if centrally located. If the hemorrhage be extensive, the sight is immediately lost, and fundus examination is impossible. The blood becomes absorbed, leaving numerous fixed or floating opacities. (Figs. 207 and 208.)

FIG. 207.

FIG. 208.

FIG. 207.—Recent hemorrhage and exudation into vitreous, following penetrating wound of ciliary region. (Photographed from specimen.)

FIG. 208.—Organized exudation and membranes in vitreous, following iridocyclitis from penetrating wound of ciliary region. (Photographed from specimen.)

Treatment. Mercurial preparations, iodide of potassium, pilocarpine, saline mineral waters, ergot, artificial leech on the mastoid, and, for the first day or two, cold applications, followed later by hot compressing.

Entozoa in the Vitreous. The scolex of pork measles, Cysticercus cellulosæ, and of beef measles, cysticercus of Tænia mediocanellata, have been occasionally found in the eye, more frequently in Germany, where it is customary to eat uncooked or improperly prepared meat.

To acquire this complaint, the patient must first develop a tapeworm in the intestinal tract; the egg entering the general circulation, is carried to the eye, and grows therein as a cysticercus. It is most commonly recognized after it enters the vitreous, being usually first deposited under the retina. The Filaria sanguinis hominis and

the echinococcus (the youthful stage of the tapeworm in the dog) have likewise been found, but are of more rare occurrence.

Treatment. Prophylaxis is most important. Food should be well cooked, and pet animals not allowed to lick the hands. Attempts have been made to extract cysticerci from the vitreous, but none as yet have resulted in restoration of vision; enucleation of the eyeball is therefore indicated.

CHAPTER VIII.

SYMPATHETIC OPHTHALMIA.

By H. GIFFORD, M.D.

When an eye is painful or irritated from any cause, it is liable to set up a sympathetic irritation in the other eye, the symptoms consisting in more or less photophobia and lacrymation, sometimes with slight ciliary congestion, or simply in an inability to use the eye steadily either for near or distant vision. This sympathetic irritation must be sharply distinguished from sympathetic ophthalmia. It may, and frequently does, exist for many years without any permanent injury therefrom, and it disappears promptly when the other eye or the source of irritation in it is removed. This irritation is simply a reflex from one eye to the vasomotors of the other, and although in former years it was believed that such reflexes could cause plastic inflammation, and a certain amount of experimental evidence was produced in favor of the idea, it is now generally conceded that reflex irritation, if it acts at all in the production of a genuine inflammation, can do so only as a predisposing cause, the presence of some chemical irritant, generally produced by micro-organisms of some kind, being necessary to complete the process.

Sympathetic ophthalmia is a plastic inflammation, generally involving the whole uveal tract, occurring in the vast majority of cases after a penetrating wound of the other eye. It matters not how extensive a wound may be, if it heals promptly, without symptoms of infection, sympathetic ophthalmia rarely or never results from it. But however slight the wound, if it is followed by a lingering congestion and irritation, the possibility of sympathetic ophthalmia must be taken into account. On the other hand, where an eye is severely injured, without any penetrating wound, long-continued congestion is quite common, but sympathetic ophthalmia rarely results. A few cases are on record where it has followed subconjunctival ruptures of the sclera, with or without luxation of the lens under the conjunctiva, also rarely as the result of ossification of the choroid, and, rarest of all, from traumatic detachment of the retina. A perforating corneal ulcer sometimes causes sympathetic ophthalmia, most commonly where a large defect has been produced, with subsequent entanglement of iris tissue in the scar. Tattooing such scars has also caused sympathetic ophthalmia. A great number of other causes of sympathetic ophthalmia which have been described are the result mainly of the want of

discrimination between sympathetic ophthalmia and sympathetic irritation.

Before the rôle played by micro-organisms in the production of inflammation was understood, it was natural that the ciliary nerve theory of von Graefe, according to which sympathetic ophthalmia is simply the result of the reflex irritation from one eye to the other, should generally be accepted. It was also natural that with the advent of bacteriology the almost constant connection between sympathetic ophthalmia and penetrating wounds of the eye should suggest that micro-organisms were the cause of the disease, and, after the apparently convincing experiments of Deutschmann, which seemed to demonstrate the easy passage of bacteria from the interior of a wounded eye along the sheaths and lymph spaces of the optic nerve to the chiasm, and thence down between the sheaths of the other optic nerve to the fellow eye, this view rapidly displaced the ciliary nerve theory. These experiments, however, did not receive general confirmation. The great majority of subsequent investigators failed to produce anything like sympathetic ophthalmia in animals, and it is only in exceptional cases that micro-organisms have been found in eyes which have been enucleated on account of causing sympathetic ophthalmia. Nevertheless, it is commonly conceded that such well-marked inflammation as that which usually occurs in sympathetic ophthalmia can hardly result from anything but the growth of micro-organisms which reach the second eye from the first, either through the lymph or blood channels, the congestion caused by the sympathetic irritation possibly causing a focus of lessened resistance in the second eye, thus favoring the lodgement and growth of germs therein. The most ardent advocates of the germ theory of the disease admit, on their part, that the nature of the germ which causes the disease, and the path which it takes in reaching the second eye, are as yet unknown, although the free communication between the eyes by way of the lymph spaces surrounding the optic nerves suggests this as the easiest and most probable route, the main objection to it being the non-occurrence of serious brain symptoms in sympathetic ophthalmia. These would naturally be expected if the germs have to pass through the cranial cavity on the way to the second eye. This objection is met, to some extent, by the suggestion that there is probably only a very slender stream of germs passing from the first eye to the second, and that these may produce serious symptoms only where they accumulate in the terminal lymph spaces of the second eye. It has, moreover, been noted that quite severe headache is not an infrequent accompaniment of sympathetic ophthalmia, and, in a few cases, the victims of the disease have become deaf as well as blind.

The rare cases in which sympathetic ophthalmia has followed subconjunctival rupture of the sclera, intra-ocular tumors, and other conditions in which the eyeball has apparently not been opened for the entrance of the germs, call for some explanation. They all have

this in common, that the exciting eye, although without apparently having been opened, is the seat of an active inflammation, probably of an infectious character, the germs in the cases of subconjunctival rupture probably having obtained entrance through minute ruptures of the conjunctiva, while in the cases from intra-ocular tumors and ossification of the choroid the infection of the first eye is probably either from the blood, or is a revival of some old infection, most of the eyes with ossification having been injured many years before. A similar explanation applies to the cases in which a sightless stump remains quiet and harmless for many years after the original injury, but becomes inflamed and excites sympathetic ophthalmia upon receiving a bruise, or when the patient catches cold or has some general infection. Cases of this kind have been reported after measles and mumps.

Formerly much stress was laid upon the special danger of wounds in the ciliary region, and this was supposed to give important testimony in favor of the ciliary nerve theory. Granting the premise, it can be explained more satisfactorily on the germ theory. Such wounds are among the commonest of penetrating injuries; they are complicated generally with prolapses of iris or choroidal tissue, which are well-recognized factors in favoring endocular infection; and, finally, they lead into the soft tissue of the ciliary body which has been found to be an especially favorable breeding-ground for various germs.

The dictum laid down years ago, that eyes in which panophthalmitis has developed never cause sympathetic ophthalmia, has been shown to be incorrect, although it is probable that sympathetic ophthalmia is less common after a violent destructive inflammation than after a milder and more chronic form, this being due probably, in part, to blocking up and destruction of the lymph channels leading from the eye, and possibly, also, to destruction of the hypothetical germ of sympathetic ophthalmia by the rapid growth of the pus germs which are generally found in these cases.

Regarding the length of time which elapses between the original injury and the outbreak of sympathetic ophthalmia, it may be said that the most dangerous period is from three to eight weeks after the injury, although a few doubtful cases have been reported within from one to two weeks after the injury, and some well-authenticated ones as early as two weeks thereafter. At the other end of the scale there is no time limit; cases have occurred forty years after the original injury, although always in the well-authenticated cases after a recrudescence of an old inflammation.

Symptoms. Where the patient is old and intelligent enough to give accurate testimony, the first symptom of sympathetic ophthalmia in most, if not all cases, is a slight failure of vision. Almost coincident with this there occur very slight signs of incipient iritis in the form of ciliary congestion (hardly noticeable in some cases), with minute spots of deposit on the posterior surface of the cornea

or the anterior surface of the lens, these latter being hardly visible except by strong magnification. Then, in the severer cases, follow rapidly adhesions between the iris and lens capsule, increase of ciliary congestion, turbidity of the aqueous, discoloration of the iris; in short, all the symptoms of plastic iritis, and, in rare cases, hypopyon. The indications of sympathetic irritation, photophobia, and so forth, which were formerly much relied upon as warnings of the approach of sympathetic ophthalmia, are generally conspicuous by their absence, and the pain is seldom great, except in the later stages. In the few cases which have been seen sufficiently early to permit a careful examination of the fundus, slight optic neuritis has often been seen, and in some cases the main symptom of the disease has been a well-marked inflammation of the optic disk. But ordinarily the vitreous becomes turbid so rapidly that the fundus is never distinctly seen. In a few eyes which have cleared up after severe sympathetic ophthalmia, small roundish spots of choroidal atrophy have been noted by different observers. All grades of severity of the inflammation occur; in some cases it never goes beyond what would be called a mild serous iritis, and yields readily to appropriate treatment. These mild cases are, unfortunately, exceptional; as a rule, in spite of all treatment, the disease progresses steadily, the iris, in spite of unlimited atropine, becoming adherent to the lens, often not only at the pupil-margin, but over the greater part of its posterior surface. The ciliary congestion continues for months or at intervals for years, the nutrition of the lens being interfered with to such an extent that it generally becomes opaque; and, while some eyes go through a stage of secondary glaucoma, the end in the majority is a mild form of phthisis. There is nothing about the appearance of the eye, in sympathetic ophthalmia, to distinguish it from any severe iridocyclitis, although, since we perhaps see more eyes that have been blinded by sympathetic ophthalmia than by any other form of plastic uveitis, the atrophic discolored iris, the shallow anterior chamber, and the grayish immovable pupil, which result from any severe and long-continued inflammation of the uveal tract, become associated in our minds with sympathetic ophthalmia.

Pathology. Comparatively few such eyes have been examined with the microscope, the bulk of what has been written about the pathology of sympathetic ophthalmia referring to the eye which has caused the inflammation, and not to the sympathizing eye. In those which have been recorded, the entire uveal tract has been found to be the seat of an active inflammation, with numerous accumulations of leucocytes such as, in a progressive inflammation, pathologists believe to indicate the presence of micro-organisms, the same extending for some distance back into the optic nerve and its sheaths. In the few cases in which both eyes have been obtained from the same subject the changes have been strikingly similar in each eye. One of these presented the unusual occurrence of so large a number of giant cells both in the uveal tracts and in the optic nerves that

tuberculosis was thought of, but no tubercle bacilli could be found, nor did the inoculation of rabbits indicate their presence. As a rule, no micro-organisms have been found either in the injured or the sympathizing eyes; but in one case they were found in both eyes, along both optic nerves, and in front of the chiasma. This case is generally regarded with some suspicion that there may have been a general infection. On the whole it may be said that the pathological findings confirm the impression given by the clinical history, that sympathetic ophthalmia must be the result of germ growth in the tissues, but that our technique at present does not permit the detection of the germ.

Prophylaxis. After sympathetic ophthalmia has broken out, the results of the treatment are, as a rule, so unsatisfactory that special stress must be laid upon the prophylaxis. To be as effective as possible, this must begin with the prevention of the wounds which commonly cause the disease. This is too broad a subject to be discussed fully here, but the importance of keeping sharp instruments out of the hands of children, and of encouraging workmen engaged in pounding metal or stone to wear protective glasses, may be mentioned. Next in order is the proper management of perforating wounds and of foreign bodies in the eye. This will be considered later on. The problem for immediate solution is: given a perforated eye with symptoms of infection which fail to yield promptly, how long shall operative interference for the sake of the other eye be deferred, and if such interference is decided upon what shall be done? Where the wound is extensive and the sight is irrevocably lost, evisceration should be done as soon as it is evident that healing is not going to occur without much reaction. Many such eyes will become quiet and do no harm to the other eye; but where no useful sight is possible the most sensible plan is not to take the slightest risk of danger to the other eye. Where there is a prospect of the injured eye retaining useful sight, antiphlogistic measures should be persevered in for at least ten days; and then if no decided improvement in the inflammation is apparent, it should be explained to the patient or his relatives that there is some danger which can only be averted with the utmost certainty by sacrificing the injured eye. But if he is willing to assume the slight risk, the operation need not be urged strongly for another week. Then, if the symptoms of infection still persist undiminished, the sacrifice of the injured eye should be urged more strongly, always with the proviso that there is no certainty of the uninjured eye becoming infected, even if no operation is done. The risk is not so very great, but the patient must understand that it will continue as long as the injured eye is at all inflamed. If an evisceration will not be consented to, an opticociliary neurectomy may be suggested; and if this also is refused, the physician, having shifted all responsibility for possible injury to the good eye, should persist in keeping the patient at rest and in following up the antiphlogistic measures as long as symptoms of irritation

continue. In the case of blind eyes which are entirely free from symptoms of deep infection, no interference is called for, in my opinion, except for cosmetic purposes; but in the case of a sightless eye which is the seat of recurrent deep-seated inflammation, the patient should be warned of the possibility of danger, and evisceration advised even if the blindness was not originally caused by an injury. In advising the patient of the danger of sympathetic ophthalmia, great emphasis should be laid upon the fact that it comes, as a rule, without warning, and that after it once appears all treatment may be in vain; and in watching for its appearance physician and patient should pay special attention to the slightest diminution of sight, daily tests under uniform conditions of illumination being made. As a purely prophylactic measure I always prefer evisceration to any other operation.

It should be understood that neither opticociliary neurectomy, nor evisceration, nor enucleation is an absolute protection against sympathetic ophthalmia. The disease has been known to occur after all of these operations, appearing after a period varying from one to fifty-four days subsequent to the operation. Some of these after-cases are difficult to explain upon any theory, but they are probably due to the infection having spread for some distance back of the eye before the operation was performed. When sympathetic ophthalmia has already appeared, the treatment varies according to the amount of sight in the other eye and the length of time which has elapsed since the first symptoms. If seen within a day or two after the first onset of the disease, I should advise immediate enucleation of the other eye, even if it has useful sight. But if not seen for several days or weeks after the first symptoms, and the first eye possess fair sight or has a prospect of obtaining it by a cataract extraction or some other operation, it is probably better not to sacrifice it; but if it have only a little sight, and there is no prospect of its ever having more than enough barely to allow the patient to get around, I should sacrifice it, even in the later stages of the disease, if the second eye still had the better sight of the two. In this I differ from the majority of writers, who follow the rule never to enucleate after an outbreak of sympathetic ophthalmia, if the first eye have or can have useful sight. My reason for this is that the statistics indicate as plainly as possible that the second eye has a better chance when the first is enucleated, even long after the outbreak of the disease. The case often cited in which, where an enucleation was recommended and refused, the condemned eye retained useful sight while the other became entirely blind, should have little weight, because if the enucleation had been consented to, it might have saved much better sight in the second eye than eventually was retained by the first. The effect of an enucleation is sometimes so prompt, a badly inflamed eye often showing most marked improvement on the day following the enucleation, that it seems probable that the first eye exercises a constant influence on the inflammation

in the second, either by reflex irritation or by the passage of toxins. (It has been shown conclusively in rabbits that toxins readily pass from one eye to the other, causing marked inflammation there without any decided meningitis or general disturbance.) This sudden improvement following enucleation is, to be sure, generally of short duration in severe cases, but in the long run the comparison of a series of cases in which enucleation has been performed with another in which it has not, speaks plainly in favor of the operation. Enucleation is recommended here in place of any other operation, because it takes out more of the infected tissue, especially if the nerve is cut far back, as it should be in such cases. It is not certain that enucleation gives better results than evisceration would, but where the outlook is so bad at the best, cosmetic considerations, which are the main argument in favor of evisceration, should have no weight as against even a theoretical argument in favor of something else.

Treatment other than surgical should consist in rest, the free use of atropine, and large doses of salicylate of sodium, mercury, iodide of potassium, or quinine. I mention the salicylate first because it is the only remedy which I have known to exert a marked effect upon a severe case of sympathetic ophthalmia. In two very severe cases in which I have used it normal vision was restored in one, and useful vision in the other, while in a third case, well marked but not so severe, normal vision was restored. By large doses I mean 10 to 13 grains in the course of sixteen to eighteen hours for each ten pounds of the patient's weight. That is, a man of 150 pounds would receive from 150 to 200 grains in the course of the waking hours. These amounts are borne best when given in brandy, 15 grains to the teaspoonful, followed by a quarter of a glass of water; but if this nauseates the patient, it may be given in capsules, brandy and water being taken separately. Some patients may not stand such large amounts, and if there is any question of heart trouble, one should begin with smaller doses. Full doses can generally be borne for two days out of three. In cases where the stomach rebels the remedy may be given by the rectum. If it has the desired effect, it should be continued, with increasing intervals of entire abstinence from it (a large dose on two days in a week is better than half the amount on four days), until long after the last sign of congestion has disappeared; and this rule applies to whatever form of medication is employed, on account of the danger of relapses.

Mercury is best given in the form of inunctions, a piece the size of the patient's whole thumb being rubbed in twice a day for four days in succession, unless tenderness of the gums appears sooner; then the same amount once a day for the rest of the week, after which an interval of several days should occur before the inunctions are recommenced. If salicylate or mercury have no marked effect, large doses of iodide of potassium or quinine should be tried, or they may be used in the intervals when the patient is not using

the other remedies. If this is done, it would probably be best to avoid following salicylate with quinine, on account of the effect on the ears.

As a rule, hot applications have a favorable effect, though in some of my patients, when used in the ordinary way, they have seemed to do harm, while when employed in the form of thick soft poultices, changed every ten minutes for an hour, four times a day, the effect has been decidedly beneficial.

If other remedies fail, subconjunctival injections of two or three drops of sublimate, 1:1000, or ten drops of 2 per cent. sodium chloride every third day may be tried. If the first eye be retained, it should receive the same local treatment as the other eye as long as signs of infection continue.

It goes without saying that everything consistent with rest which can be done to keep the general condition good should be done. In spite of all treatment, even when the case is seen at the start, the prognosis is bad. The iris generally adheres closely to the lens in spite of all the atropine that can be borne, and, besides the blocking of the pupil with exudate, sight is reduced still further by opacities in the vitreous and lens. Where glaucoma ensues an early operation for its relief may be required; in these cases the iris commonly bulges at the periphery, and a double transfixion of it with a cataract knife should be tried before resorting to an iridectomy. But, except in the case of glaucoma, no operation should be done for the improvement of sight until all signs of active infection have been absent for a year. Even then the results of operations are apt to be discouraging. Good light sense and projection are retained surprisingly long in these eyes, and to a novice the task of restoring sight by an iridectomy or extraction may seem simple enough; but when an iridectomy is attempted, it is generally found that only the anterior layers of the iris can be removed, the pigment layer remaining to block effectually the artificial pupil. If the lens is extracted, the sight generally still remains poor, on account of extensive vitreous opacities, and any operative interference is apt to produce a marked reaction, accompanied by the production of exudate which occludes the new pupil, so that repeated subsequent iridotomies or excisions of the inflammatory membranes with de Wecker's scissors have to be resorted to. All these repeated attempts should be made at considerable intervals, and in a fair proportion of cases patience will be rewarded by a reasonable amount of success.

In many cases no chance for operative interference is ever given, the inflammation continuing until all sight is lost, while it sometimes happens that in addition to blindness the pain becomes so intolerable that enucleation or evisceration of the second eye has to be performed.

The gloomy picture hitherto presented applies to the severer cases, and these, up to the present time, constitute a large majority of those described; but it is evident that of late more favorable reports

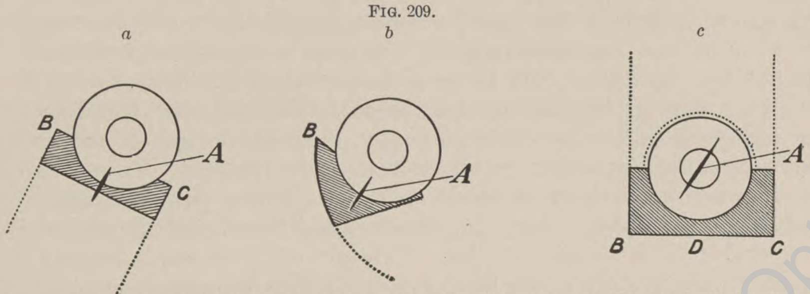
are being received. This is perhaps due less to improvements in treatment than to a more widespread knowledge and an earlier detection of the disease. Moreover, it is probable that mild forms of sympathetic ophthalmia are much more common than formerly was supposed. I have known it to develop and run its course in two cataract patients with so little congestion or subjective disturbance of any kind that, as the sight was already obscured by the cataracts, the patients were unaware that any inflammation had occurred. Where sympathetic ophthalmia has occurred after enucleation or evisceration, the course has almost invariably been mild and fair or perfect results have been obtained. The same is true to some extent of the cases which have occurred in connection with ossification of the choroid.

The Treatment of Penetrating Wounds of the Eyeball. Where a clean-cut penetrating wound of the eye, without any prolapse of iris, choroid, or vitreous has occurred some days before the patient is seen, the fate of the eye, as far as the infection is concerned, is generally decided beforehand. If the eyeball shows little or no congestion, except in the neighborhood of the wound, and no pain is experienced, it is generally safe to conclude that infection has been escaped, and all that is immediately required, unless secondary glaucoma is present, is to protect the eye, preferably with some form of shield bandage, use sufficient atropine to keep the iris from adhering to the lens, and keep the patient quiet. Of course, if a traumatic cataract is present, it may be extracted, if there is no increase in the secretions; but if there is, it is better to put off the extraction for a week or so, treating the lids with some astringent meanwhile.

When, however, a fresh penetrating wound is seen, the treatment should begin, wherever possible, with a thorough irrigation of the conjunctival sac and surface of the globe with a sterilized 0.5 per cent. solution of salt or some other sterile non-irritating solution. Then, after the excision of any prolapsed iris or vitreous, should follow the protection of the wound with a conjunctival flap; for it should be realized that nearly every conjunctival sac contains more or less pathogenic germs which cannot be thoroughly cleared out by any measures, chemical or mechanical, which it is safe to use; and while in some cases of infection the germs may be present on the offending substance before it reaches the eye, the probability is that in nearly all cases the germs either are carried in from the surface of the globe at the time of the injury, or they gain entrance from the conjunctival sac after the injury has been inflicted. To prevent this latter contingency there is no protection equal to that afforded by a conjunctival flap. Where the wound is entirely in the sclera the best plan is to excise a triangular bit of conjunctiva at one side of the wound, and draw a triangular flap over this raw surface from the opposite side of the wound by one or more sutures. Where the wound extends a short way into the cornea the simplest plan is to dissect up the conjunctiva all around the cornea and draw it over the whole cornea

by a purse-string suture tightly tied. A more complicated but on some accounts better plan—as it allows the physician to keep a better watch of the condition of the iris, and as permitting a better action of atropine—is to excise a portion of the conjunctiva at both sides of the wound, as indicated in Fig. 209, *a*, and to draw the loosened conjunctiva over these raw surfaces and the wound at the same time by two sutures, or, as in Fig. 209, *b*, by a single suture. Where the wound involves the central portions of the cornea, it may be that the purse-string suture will offer a sufficient protection, but a more certain plan is to excise a portion of the conjunctiva all around the opposite half of the cornea, as in Fig. 209, *c*, and bring a conjunctival flap from above clear across the cornea. It may be questioned whether the use of a protecting flap is necessary in the case of clean-cut wounds of the cornea; but where the wound is irregular or contains iris tissue or fragments of broken-down lens or vitreous, there can be no doubt about its advisability. In the case of wounds

FIG. 209.



The shaded areas indicate the surface from which the conjunctiva should be excised; the dotted lines, the outlines of the conjunctival flaps. *A*, wound to be covered; *B*, *C*, *D*, points where sutures are to be applied in drawing the flaps into position.

that are not above suspicion the edges should be touched with the galvanocautery or Paquelin cautery, or, if these are not at hand, with a chemical caustic, such as carbolic or nitric acid, applied with the utmost care with a very finely drawn-out cotton swab, before drawing over the flap. To be logical, one should use the same line of treatment for operative wounds where vitreous is lost or iris tissue is caught in the wound. And it is certain that if the wounds had been protected by a conjunctival flap many and probably all of the cases of sympathetic ophthalmia which have been reported after cataract extraction could have been prevented. The small conjunctival flap, which is often made as the final step in the incision for cataract extraction is good as far as it goes, but it is seldom large enough to cover fully the large prolapses which sometimes occur after simple extraction, and if an iridectomy is made the points at which the iris is most apt to be entangled, namely, the angles of the wound, are left unprotected. To make an effective flap for protecting an extraction or iridectomy wound, the conjunctiva should be slightly raised by the injection under it of a cocaine solution at

the point of puncture and counter-puncture, and the knife entered at some distance outside of the cornea and passed along under the conjunctiva before entering the anterior chamber; then in making the counter-puncture care should be taken to pass the knife along beneath the conjunctiva as far as possible before piercing it. In cases where prolapse of vitreous is expected, two sutures should be put in at the upper part of the conjunctival wound, and drawn well out of the way before the extraction is completed. This, it seems to me, is the simplest way to protect completely an extraction wound, but it may be that to prepare a flap above the cornea and bring it down and fix it in the manner indicated in Fig. 209, *a*, or even to use a purse-string suture, will prove to be better. Some such form of extraction, although too complicated to become popular at once, is certain, after the technique is mastered, to give a better guarantee of an immediate good result and of safety from sympathetic ophthalmia.

After protecting the wound as well as possible from infection, a protective bandage (both eyes being closed for at least a day or two in severe injuries), rest, and atropine are all that will be required if no infection has taken place. It may be questioned whether it is not best to apply cold in some form during the first twenty-four hours. This is recommended by good authorities, but the difficulty of applying cold in any efficient way, without danger of disturbing and infecting the wound, is such that, on the whole, I believe as good results will be obtained without it, unless decided pain indicates that infection has taken place, in which case the continued use of ice, preferably with a light rubber or metal coil or a small ice-bag frequently filled with small bits of ice, should be employed until the end of the first twenty-four hours, the eye being protected by a pad of absorbent cotton which is kept wet with sublimate, 1:5000. Later than this I prefer the use of hot applications for half an hour to an hour three or four times a day, where symptoms of infection continue.

In all cases where infection is suspected or feared, beside the use of cold or heat and rest, atropine should be used with extra liberality, 10 to 15 drops of a 1 per cent. solution being instilled and allowed to run out at the outer angle of the eye two or three times a day, and large doses of sodium salicylate should be given. Subconjunctival injections of 1 to 3 drops of sublimate, 1:1000, or 6 drops of cyanide of mercury, 1:2000, or 10 drops of sodium chloride, 2 per cent., may be tried every second or third day. In some cases their effect is astonishingly good; in others, for no apparent reason, they do no good, or even do harm. Where the infection is superficial, as in some cataract wounds or injuries of the cornea, a thorough application of the galvano or Paquelin cautery often does excellent service, and if neither of these is available, scraping, followed by the application of tincture of iodine or nitric or carbolic acid, will sometimes be as effectual. In desperate cases, where it is evident that purulent inflammation of the vitreous has started, the endocular

use of the cautery has been reported to give good results; the tip of the galvanocautery having been plunged deeply into the vitreous as near the focus of infection as possible, and the current turned on while the point is moved slowly about. I have had no experience with this method of treating the vitreous, but have tried it in the anterior chamber with a bad result. If it is tried in the vitreous, the wound should immediately afterward be covered with a conjunctival flap. In treating prolapses of the iris much discrimination is necessary in the use of the cautery. Except in plainly infected cases, it is best not to use it if the iris can either be replaced or seized with the forceps and cut off. Where this is not possible, as is frequently the case with a prolapse of more than a day's standing, or where the prolapse is apparently infected, it may be used if the burnt tissue be immediately scraped away and the spot well covered with a conjunctival flap. To burn a prolapse, however, without immediately protecting the spot from secondary infection is, I believe, distinctly dangerous. It is often done with good results, but evidence is constantly accumulating to show that it has a decided tendency to favor the occurrence of sympathetic ophthalmia.

Where a small prolapse is seen several days after the occurrence of the injury, and is evidently not infected, it is perhaps best to leave it entirely alone, unless the physician has had experience in the technique of applying conjunctival flaps. The majority of such prolapses subside quietly and are abundantly protected by new-formed connective tissue if the eye is kept quiet and hot applications are used.

The ideal treatment of iris prolapse is to replace it, and thus restore the form of the pupil; but this seldom is done after accidental wounds, partly because adhesions form so quickly that unless the eye is seen soon after the injury the iris cannot be replaced without tearing it; and partly because when replacement is attempted in the ordinary way, the replaced iris is immediately pushed back into the wound as the instrument used is withdrawn. A plan which gives better results, especially if the wound has not reached the extreme periphery of the cornea, is to make a new incision at some distance from the wound just sufficiently large to permit the introduction of a small spatula, with which the prolapsed iris can be drawn back into the anterior chamber, and as the first wound closes behind it there is little tendency for it to be again expelled.

Management of Foreign Bodies in the Eye. Where a foreign body has entered the eye the management depends upon the presence or absence of infection and the nature of the foreign body. Where a piece of iron or steel has entered, its removal should be attempted at once with some form of magnet. Other foreign bodies, except when in the lens, must naturally be removed with forceps or hooks, if they are to be removed at all. Sometimes their removal is not necessary. Pieces of wood, stone, lead, copper, and glass may be tolerated in the eye for an indefinite period if no infection has occurred at the

time of their entrance. So that while, in a recent case, if such a foreign body can easily be seen and reached, its removal should be attempted at once, it should be left alone if this is not the case and there are no symptoms of infection nor other serious disturbance, since the attempt to remove it in the dark, even with the best localization possible by means of the x-rays, is more likely to do harm than good unless it be very large. The amount of toleration which the eye exhibits toward aseptic foreign bodies which have entered it without carrying in or being followed by germs from the conjunctival sac, depends primarily upon the amount of chemical reaction which occurs between them and the fluids of the tissues; secondarily, upon their becoming firmly fixed by fibrous exudate. Copper, iron, zinc, and lead all are acted upon so as to produce irritating compounds, copper most markedly, lead least of all. Copper is the only one which, without the aid of germs, will produce a purulent exudate. It also has the property of producing a softening effect upon the tissues, so that in quite a number of cases, if left to itself, it will work its way to the surface and be expelled spontaneously. This has been known to happen after a lapse of twenty-one years. In rare cases a bit of copper is encapsulated so completely that its chemical action appears to cease and it causes no disturbance after the first reaction has subsided. It is important to remember the chemical activity of copper in the eye, because a moderate amount of reaction, even when long continued, need not cause the same anxiety that would be natural if the inflammation were thought to be the result of infection. The copper may be merely working its way to the surface. It is not best, however, for the physician to assume all the responsibility of advising against operation in such cases. Whenever the reaction is prolonged, there is a possibility of sympathetic ophthalmia, which can only be obviated by removing the foreign body or sacrificing the eye; and if it is decided to attempt to remove a bit of copper which has been in the eye for some time, it should be remembered that, however accurately it may have been located at the time of its entrance, it is liable to be found at some distance from this point later on. Lead occurs in the eye chiefly or exclusively as the result of shot wounds. These wounds are peculiar in that in spite of being so small, unless the shot is going with sufficient rapidity to go clear through the globe, the impact of the blunt though small object is sufficient to produce so much internal disturbance in the form of hemorrhages or detachments that the sight is lost or very seriously injured, even when no sepsis occurs. Although the wounds produced by shot in the external tunics are so small that they are generally left to themselves, fresh ones should, I believe, always be protected by a conjunctival flap drawn far beyond their limits in the manner previously indicated. This having been done, I consider that any attempt to remove the shot, unless it can actually be seen with the naked eye, is a mistake. If aseptic, as they usually are, shot will generally become encapsulated and cause no subsequent irritation. These injuries generally cause

so much tissue disturbance that the congestion is apt to be prolonged, even where there is no sepsis, and if, some days after the injury, the anterior chamber fills up with blood from the vitreous, it is well to perform a paracentesis, even repeatedly, to help clear up the vitreous. Shots that go clear through the ball into the orbit need no attention, though they sometimes cause complete blindness by injuring the optic nerve, either directly or by the pressure from the hemorrhage which they cause. But even where the sight is immediately and completely lost after such an injury, an absolutely bad prognosis should not be given at once, because at least one such case is on record where the sight returned, probably because the blindness was due to pressure from hemorrhage rather than from direct injury to the nerve.

The diagnosis of shot in the eye should not be made too hastily. Occasionally a shot strikes the eye obliquely, causing a hemorrhage at the margin of the cornea and a rupture of the iris with hemorrhage in the anterior chamber without any penetration, although at first glance the physician is inclined to believe that the shot must be in the eye.

Other metals, except iron or steel, which will be discussed later on, occur so rarely in the eye that they hardly need to be considered.

Small pieces of wood, glass, stone, and other indifferent substances are often retained indefinitely without disturbance, if they remain immovable; but if free, they often cause a mechanical irritation which necessitates an attempt to remove them. In deciding upon such an operation the danger of mistaking, for the foreign body, a bit of exudate on the iris or lens should be kept in mind. Such a mistake is sometimes impossible to avoid, and it is probably often made.

A small foreign body in the lens without infection is best left alone until the lens is opaque, when it will generally come out with the latter, especially if a broad incision and an iridectomy are made.

Eyelashes are not infrequently carried into the anterior chamber or vitreous; they often cause no disturbance; but in the anterior chamber they apparently sometimes form the starting point for cysts, and in other cases the germs which commonly adhere to their roots may cause the loss of the eye.

The Management of Bits of Iron or Steel in the Eye. The management of bits of iron or steel in the eye deserves to be considered separately, both on account of their behavior in the eye and from the fact that, unlike all other foreign bodies, a large proportion of them can be removed without their being visible beforehand. Although a fair proportion of them, if aseptic, become encapsulated with so little reaction that the impression is given that no further harm will be done by their presence, sooner or later they almost invariably become decomposed, and the soluble compounds thus formed are disseminated through the eye, producing the condition known as *siderosis*, which shows itself externally by a brown discoloration of the iris. Eyes in which this decomposition has gone on for any

length of time almost always, through disorganization of the vitreous and detachment of the retina, lose any sight that may have been left, and frequently become so irritable that they have to be sacrificed, both for the sake of comfort and to avoid the danger of sympathetic ophthalmia. As an example of the danger of allowing apparently innocent bits of steel to remain unmolested, the following history may be useful. A young man was brought to me shortly after a bit of steel, not larger than half the head of a pin, had penetrated his cornea, iris, lens, and retina, and remained so firmly fixed in the choroid and sclera that the giant magnet did not move it. As the opacity first present in the lens cleared up almost entirely and the vision became nearly normal, the eye being entirely free from irritation, it was thought best not to interfere with it. This condition remained unchanged for several months, when the sight began to diminish and the eye became irritable. He did not return, however, for nearly a year, and when he did the sight was entirely gone and the eye was so troublesome that it was removed. As illustrating what may be accomplished in a similar case by bolder methods, there is one case on record in which the operator with the aid of the ophthalmoscope loosened the bit of steel with a discission needle passed through the sclera, and then with the giant magnet drew it into the anterior chamber, whence it was easily removed.

Where the piece of metal is very small it sometimes becomes entirely disintegrated before the sight is wholly destroyed, and in this event the siderosis sometimes clears up, and useful sight is retained without any operation to remove the metal.

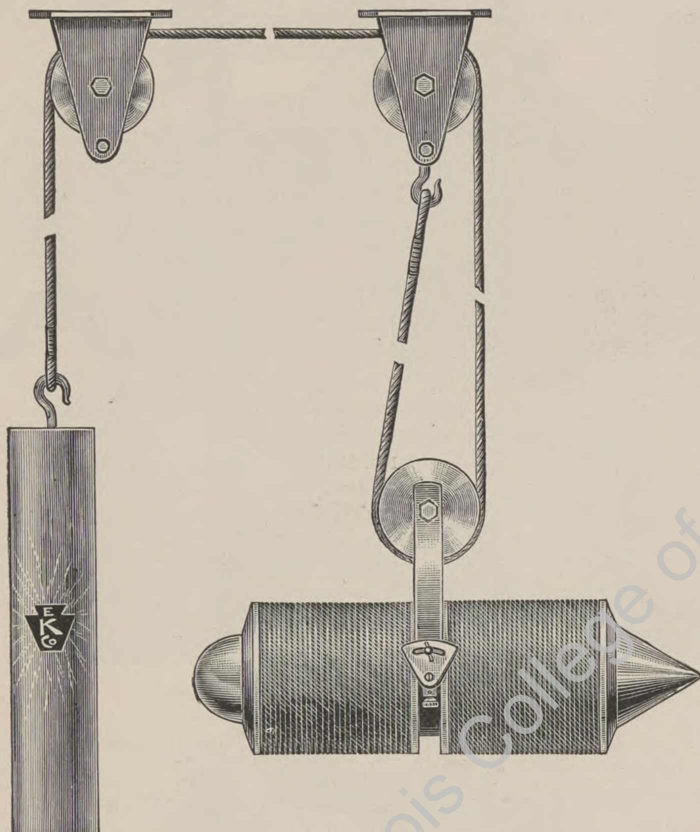
In the management of these cases the physician should have at least one electro-magnet, for while a certain number of bits of steel can be removed from the anterior chamber, the iris, lens, or even the vitreous, without a magnet, the attempt to do this will not infrequently fail, and many an eye which might otherwise be saved will be lost if no efficient magnet is at hand. If a man can have but one magnet, perhaps the most generally available form is the small one of Hirschberg or Sweet; each has several interchangeable points, of which the larger and blunter ones should be tried if there is any probability of the metal being near the surface, while the smaller ones can be passed into the anterior chamber or deep into the vitreous.

This can be operated with various kinds of galvanic batteries, but it is much more satisfactory to have it connected with some power current, and if this is to be done, the voltage of the current to be used should be mentioned in ordering the magnet. But to do the most satisfactory work in this line, it is necessary to have, in addition, a giant magnet or Haab magnet, a sideroscope, and access to an x-ray apparatus. (Fig. 210.) The Haab magnet may either be mounted on a stand or suspended from the ceiling, and while it can be operated with storage or other batteries, the connection with a power current is much more desirable. Some apparatus for turning on the current

gradually is an advantage, but much the same effect can be produced by gradually bringing the eye toward the magnet.

The sideroscope of Asmus consists essentially of a magnetic needle suspended by a fibre of silk. By attaching a mirror to this and viewing through a telescope the image of a light reflected from it upon a scale at some distance, extremely delicate results can be obtained in detecting and locating bits of steel or iron within the eye; but as the instrument requires a special room, at some distance from any

Fig. 210.



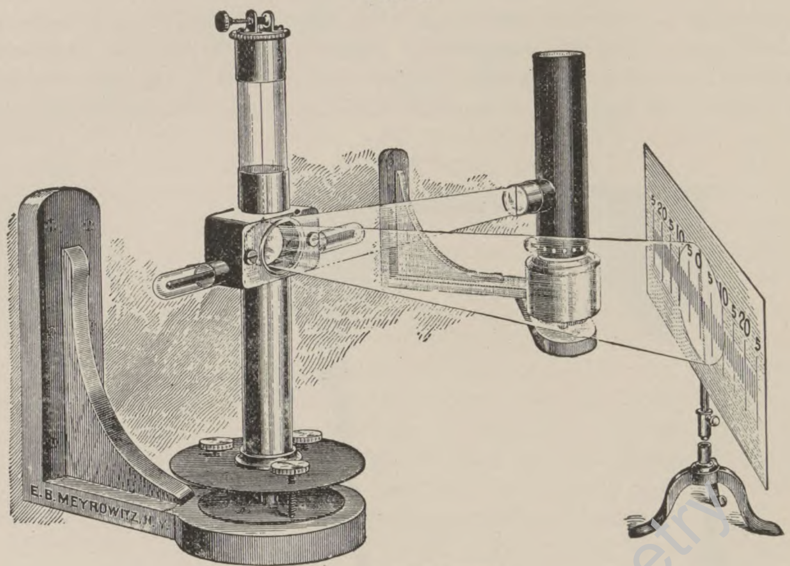
Haab's magnet

trolley wires, it will probably be used chiefly in eye hospitals. Hirschberg has had a less complicated form of the sideroscope made which may come into more general use.

The use of the x-rays in discovering and locating foreign bodies in the eye is, of course, not confined to bits of iron or steel. Their chief use is in determining whether or not any foreign body is present. If the body is large, it can frequently be seen with the fluoroscope, in which case a fair idea of its location can sometimes be had by having

the patient move his eye in different directions during the observation, the shadow moving with the cornea, if the foreign body is in

FIG. 211.



Hirschberg's sideroscope.

FIG. 212.

Sweet's apparatus for localizing foreign bodies in the eye.

the anterior half of the globe; against it, if it is in the posterior half. Much more accurate work may be done by the method of Sweet,

which, in brief, consists in taking two or more radiographs with the tube in different positions, the plates being held in exactly the same position by a special arrangement which at the same time holds two metal points against the lids at a definite distance from the plate. By comparing the positions of the shadows of these points and of the foreign body on the radiograph, and carefully plotting the paths which the x-rays must take from the tube to the plate through a circle representing the position of the eyeball, the location of the foreign body can be determined with considerable accuracy by the point at which the paths of the rays through it intersect within this circle. If Sweet's apparatus is not at hand, fair results can be obtained by fastening three shot with collodion and cotton on the outside of

FIG. 213.

Radiograph showing piece of steel in the eye. (SWEET.)

the closed lids, above, opposite, and below the cornea. It is also well to do this before fluoroscopic examinations. As an example of the advantage of having different methods of diagnosis at one's disposal, a recently reported case may be cited in which, after an injury, a piece of steel in the eye was suspected, and where the sideroscope plainly indicated the existence of such a fragment; the giant magnet, however, produced no effect upon it, and the x-rays showed the presence of a rather large foreign body in the tissues under the edge of the orbit. The patient then remembered that he had been struck in this locality with a piece of steel a long time before.

Assuming then that the physician has all the desirable instruments for the locating and extracting of bits of iron and steel in the eye, it should be realized, in deciding what cases it is worth while to use

them on, that many patients have foreign bodies in the eye without knowing it. Many cases of otherwise unexplained one-sided cataract in young people are due to unobserved or long-forgotten injuries which sometimes have included the entrance of foreign bodies. The existence of siderosis will, of course, always suggest the presence of iron or steel, even where there is no history of an injury. In many fresh cases the history given by the patient frequently seems directly to contraindicate the entrance of a foreign body into the eye. The patient with only a small or an unrecognizable wound or scar feels sure that he must have been struck by a large piece of metal, because the blow almost knocked him down, or he states positively that he heard the piece drop after striking the eye, and yet the examination or the subsequent history will often show that no weight should be placed upon such statements, and that in all cases of injury from flying metal or from unknown causes in the proximity of men engaged in metal or stone working a foreign body in the eye should be suspected and carefully searched for.

Some such routine as the following is desirable when examining these patients: after testing the vision the eye should be examined by oblique light, and, if the fundus can be seen, with the ophthalmoscope. If this is not possible, a careful test of the field of vision will sometimes give a clue. If the suspected foreign body is evidently small and has entered through the cornea or close to it, or if a cataract is present or is evidently forming, no time need be lost in a fresh case with the use of the x-rays or the sideroscope; but the patient, after cocainization, should be placed opposite the giant magnet, the direction of the wound canal corresponding as nearly as possible with the long axis of the magnet. The current is then gradually turned on or the patient's head is gradually brought toward the magnet, the eye being kept brightly illuminated and very closely observed meanwhile. The first sign of the existence of a piece of steel will frequently then be given by the occurrence of pain, or the iris will begin to bulge in some part of its periphery, when the relation of the eye to the axis of the magnet should be changed so as to favor drawing the piece of metal through the pupil into the anterior chamber rather than directly through the iris root. In other cases the first sign is the sudden appearance of the fragment on the posterior surface of the cornea, whence, as the current is turned off, it generally falls to the bottom of the anterior chamber, from which it can easily be removed through a peripheral incision with the small magnet, or, in some cases, with the large one. (The small magnet and the necessary instruments should previously be sterilized and kept ready at hand.) If the steel cannot be drawn into the anterior chamber, nor through the root of the iris, it may be necessary to excise a piece of the latter, in order to complete the extraction.

Where there is a fresh scleral wound, or where there is a chance of preserving the lens clear, it is best to attempt the extraction

through the reopened wound, which should be enlarged at one end by a short incision at right angles to its main direction. In some such cases the steel shoots out through the wound to the large magnet before the latter touches the eye; in others the sclera in the neighborhood of the wound may be seen to bulge, but the extraction cannot be completed without introducing the tip of the small magnet for a short distance. In all cases where the extraction is attempted through a scleral wound, the greater danger of infection should be kept in mind, and beside the ordinary precautions the wound should be thoroughly protected by a conjunctival flap. Where, in using either the large or small magnet, a gradual approach to the eye or to the suspected location of the foreign body fails to remove it, the current should be turned off and on quickly several times, in the hope that the sudden jerks thus produced may loosen it.

Where the bit of metal is probably large—*i. e.*, if the external wound is three-sixteenths of an inch long—it is perhaps best not to use the large magnet at first, since the forcible tearing out of such a fragment has in some cases caused the loss of an eye which might otherwise have been saved. In these cases and in others where the large magnet produces no result, the metal should be located as accurately as possible, and if it is supposed to be in the vitreous an angular conjunctival flap should be dissected up, using the wound for part of it if this is suitably located, and one of the blunt tips of the small magnet introduced a short way between its lips. If several closures of the circuit produce no result, a longer tip should be introduced as near as possible to the supposed location of the metal; and if this attempt fail, the tip should be moved slightly in different directions, and the opening and closing tried again before it is withdrawn. Often a click caused by the metal coming into contact with the magnet is heard on closing the circuit. If it is decided to move the point to an entirely different part of the vitreous, it is best to withdraw it and pass it in straight to the desired point, in order to avoid as much as possible disorganization of the vitreous. If three or four introductions fail to bring forth the metal, it is best to desist, and, after covering the wound with the conjunctiva, wait for further developments. Sometimes a bit of steel that eludes the first attempt can be better located and removed after the clearing up or extraction of an opaque lens. In other cases the signs of infection become so evident that evisceration is called for.

Where a piece of steel is known to be in the opaque lens, it may be questioned whether it is better to extract the latter first, with the expectation that the steel will come out with it, or to draw the metal into the anterior chamber with the magnet, and remove it before extracting the lens. The first manœuvre has always succeeded in the cases in which I have known it to be tried; but if for any reason in extracting the lens there should be a prolapse of vitreous before the metal were expelled, the necessity of then introducing the magnet would certainly involve more danger than the other plan.

Eviscleration, Enucleation, and Opticociliary Neurectomy. These are the operations most commonly employed as prophylactics and to some extent as treatment for sympathetic ophthalmia.

EVISCERATION or EXENTERATION, as originally described, consists in dissecting back the conjunctiva for a short distance all around the cornea, making a small incision through the sclera about one-eighth of an inch outside of the former; then, with blunt-pointed scissors, which should be kept between the sclera and choroid, completing the excision of the cornea, including a narrow ring of sclera; then, with a narrow blunt spatula loosening the connections between the sclera and the choroid; then, with a broad, flat, sharp-edged spoon severing the connections between the choroid and the optic nerve, and by pushing with the spoon from behind and pulling with a forceps from in front, removing in one piece choroid, retina, vitreous, lens, iris, and cornea. Any bits of choroid or retina which may have escaped this process are then scraped out, and, after irrigating the cavity, the conjunctiva is drawn together over the opening with a purse-string suture. (Fig. 214.) For obtaining pathological specimens this method is

FIG. 214.

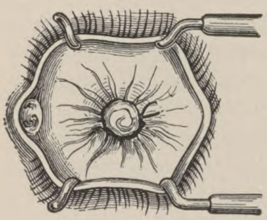


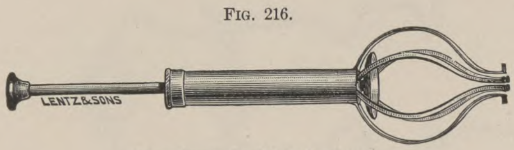
FIG. 215.

FIG. 214.—Stump after evisceration. (DE WECKER.)
FIG. 215.—Mules' vitreous spheres.

undoubtedly the best, but it is followed often by quite an extreme reaction with long-continued pain. For the comfort of the patient strictly simple evisceration—*i. e.*, without excising the cornea—is much superior. This is done by making an incision clear across the cornea, extending for a short distance into the sclera on either side, utilizing any extensive corneal wound which may be present. Through this the entire contents of the globe are scraped out with an evisceration spoon, especial attention being paid to the ciliary region and to the posterior part of the cavity, where, otherwise, fragments of choroid or retina are apt to be retained. The cavity is then filled with peroxide of hydrogen, and after this has mostly boiled out the size of the cavity is reduced by pushing in the front of the globe about half-way toward the posterior wall; iodoform or something similar is dusted freely onto the surface, some of it entering the cavity; a bit of gauze is pressed against the semicollapsed ball, the lids allowed to close over it, and a firm bandage applied. The gauze should be removed on the second day, when, if there has been no pain, the first dressing should be made, and, if any considerable pain occurs,

hot applications for half an hour from one to three times a day, continued for three or four days, will control it. The reaction following this operation is comparatively slight, and the cosmetic result is much better than where the cornea is excised. Where, as is the case with most subjects under forty years of age, the question of the after-appearance is of much importance, Mules' operation or some modification of it should be employed. The essence of this operation consists in adding to the evisceration the introduction of a glass ball into the scleral cavity. As originally performed, the cornea with a triangle of sclera at either side of it is excised by two curved incisions, the contents of the globe scooped out, a hollow glass ball introduced (Fig. 215 and 216), preferably with the aid of

FIG. 216.



Introducer for Mules' spheres.

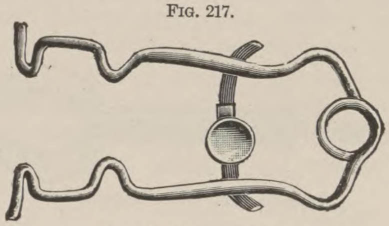
Mules' injector, the elliptical opening closed by catgut sutures, and over this the conjunctiva is brought together by silk sutures, so arranged that the line of union is at right angles to the scleral wound. This, on the average, gives an even better stump for an artificial eye than the simple evisceration. Its main drawback is that as the cavity contracts the sclera is sometimes so tightly stretched over the enclosed ball, before union of the wound is complete, that the latter reopens and the ball is extruded, sometimes months after the operation. Something may be done to lessen the danger of this by uniting the scleral wound with a continuous silk suture and drawing the conjunctiva from above down at least three-sixteenths of an inch below the scleral wound, where it is anchored by stitches passing through the episcleral tissue.

More time is given for a firm union of the wound if, instead of excising the cornea, the latter is left intact, and after dissecting up the conjunctiva around and for some distance back of it, the contents of the globe are removed through an angular scleral incision, one arm of which should be about three-quarters of an inch long, running back along the border of the superior rectus muscle, the other from one-quarter to one-half inch long, parallel to the border of the cornea, three-sixteenths of an inch back of it. This wound should be held widely open with hooks while the evisceration is being completed, and should be closed with a single silk suture at the angle, the conjunctiva being then drawn over the cornea by a purse-string suture. It is more difficult to eviscerate the eye thoroughly through such an incision, but it gives a better stump, and if the glass ball should ever be extruded from the scleral cavity after it, which I have never known to happen, it would still lie beneath the conjunctiva,

where it would probably remain. Where this operation is done, a large part of the cornea is absorbed gradually, that which is finally left being flat, insensitive, and causing no interference with wearing a glass eye. In selecting a ball for this operation, one of not more than half of the diameter of the eye should be taken, and if the cornea is excised a still smaller one is desirable.

In place of glass balls, balls of silver, with or without gold plating; aluminum, bone, catgut, sponge, and silk have been used. Some of the balls have been fenestrated or made of filigree, to catch the granulation tissue, and thus aid in preventing their extrusion. Probably none of these substances has any advantage over the glass if

FIG. 217.

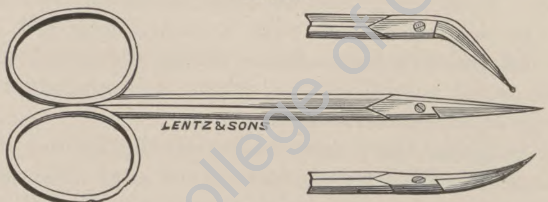


Luer's eye speculum.

FIG. 218.

Fox's fixation forceps.

FIG. 219.



Iris scissors.

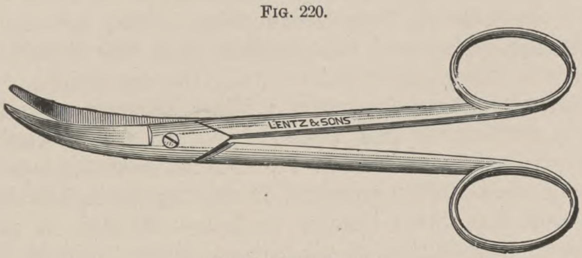
the latter is introduced properly, and some of them are distinctly inferior to it.

ENUCLEATION or shelling the eyeball out of its capsule is performed by dissecting back the conjunctiva around the cornea, cutting off each of the recti muscles in turn by means of a strabismus hook and blunt-pointed scissors, keeping the points close to the globe; then, after removing the speculum, dislocating the eyeball forward by pressing back on the lids above and below, thus making the optic nerve tense; then passing strong blunt-pointed curved scissors back along the outer side of the globe until the nerve is plainly felt, and cutting the latter far back if there is any suspicion of malignant dis-

ease, or if sympathetic ophthalmia is present, and detaching the oblique muscles as the ball is drawn forward. A pad of wet gauze or cotton is then pressed against the lids, to check the bleeding, iodoform is dusted in freely, and the eyelids closed with rather a tight bandage, plenty of cotton being used beneath it. Many operators close the conjunctival wound with a stitch or two, but I have never found this necessary nor advantageous. In cutting the rectus tendons, sufficient of the external one should be left attached to the globe to afford a point of attachment for fixation forceps. Where the operation has been preceded by long-continued or severe inflammation, Tenon's space is sometimes so nearly obliterated that the globe has to be dissected out slowly, great care being necessary to avoid perforating it.

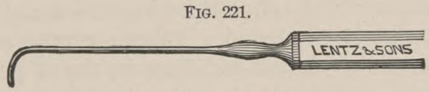
To obtain the best possible stump after enucleation, each of the rectus muscles should be secured by a catgut suture as the tendon is divided, and, after introducing a glass ball into the cavity left by

FIG. 220.



Enucleation scissors.

FIG. 221.



Strabismus hook.

the globe, the muscles should be united in pairs over its surface, the conjunctiva being brought together over them and united to them by silk sutures. Enclosing the ball in a thin layer of sterilized sponge probably favors its retention. I have had no experience with this operation, and it is too soon to say how well the glass ball is retained and tolerated after it. If retained, it certainly will give a much better result than can be obtained without it. Even where an eye has been enucleated for some time, the attempt to introduce a glass ball into the orbital tissues is justifiable, and has given, it is claimed, excellent results.

OPTICOCILIARY NEURECTOMY. This operation, which practically has superseded the previously proposed neurotomy or simple division of the nerves, consists in making a three-quarters inch vertical incision through the conjunctiva, over the insertion of the external rectus, dividing the tendon of the latter so as to leave a stump one-sixteenth of an inch long attached to the sclera, securing the

long end with a suture which is drawn well to one side, passing strong curved scissors along the globe until the optic nerve is reached and plainly felt, which can be facilitated by drawing the eyeball forward as far as possible; then dividing the optic nerve as far back as possible, rotating the posterior end of the globe forward until it can be seen, cutting off the optic nerve stump one-sixteenth of an inch back of the globe, clearing the posterior pole of the latter by curved scissors of all connective tissue and possible ciliary nerve attachments, replacing the globe, uniting the ends of the rectus tendon with two fine silk sutures, and closing the conjunctival wound. Immediately after cutting the optic nerve the lids should be closed and firm pressure made upon them with a cotton pad for at least three minutes; unless this is done, the bleeding into the orbital tissue is such that the ball sometimes cannot be replaced. For the same reason it is important to complete the operation as rapidly as possible and hold the lids closed with a firm bandage. It is sometimes necessary to sew the lids together temporarily, in order to protect the cornea. In spite of these precautions it may become necessary to enucleate the globe, and the possibility of this should always be insisted upon to the patient before doing the operation.

Choice Between These Operations; Their Dangers and Advantages. Death from meningitis has been known to follow each of these operations. Evisceration was first systematically employed to avoid the danger of death after enucleation during panophthalmitis, and, although death has been known to follow it also, it probably is less dangerous than either of the others, since there is less chance in doing it of infecting the orbital tissues. The danger is slight in any event, but its possibility should be kept in mind and the strictest precautions to avoid it observed. Whether any operation beyond freely incising the globe should be done in florid panophthalmitis is a moot point; but, on the whole, a simple evisceration, followed by the free use of peroxide of hydrogen in the cavity, is the most rational procedure, though it must be admitted that where the inflammation has been especially violent or long continued the sclera itself is occasionally so thoroughly infected that it becomes necessary to excise it later on.

Enucleation is the operation preferred by most authorities. It is a necessity in cases of intra-ocular tumors of the eye and in some cases of malignant disease of the exterior of the globe, or of the conjunctiva or orbital tissues. It should always be preferred in the treatment of an actually broken-out sympathetic ophthalmia. The main advantages in other cases are the slight reaction which generally follows it and its greater simplicity as compared with Mules' operation or opticociliary neurectomy. The dangers connected with it are, first, that of enucleating the wrong eye. It would seem almost incredible that this could ever happen were it not that it has actually occurred. A good eye has been enucleated and a blind one left. This danger is not peculiar to enucleation, for, while it

has not been known to occur with either of the other operations, there is no reason why it might not occur with them also. In some clinics it is customary to guard against this possibility by marking with pencil or ink the brow on the side to be operated upon. Death from hemorrhage has been known to occur after enucleation, also orbital cellulitis and orbital abscess, but these accidents are of the utmost rarity.

Evisceration is, in my opinion, the operation of choice as a prophylactic for sympathetic ophthalmia, although the weight of authority is in favor of enucleation. Where the simple evisceration is done, it is easier, safer, and much more likely to give a good stump than enucleation, and the reaction following it, if hot applications, or, as some prefer, ice, are employed for a few days, is little, if any, greater. The same advantages, except that of simplicity, with the additional one of giving a better stump, pertain to Mules' operation, and the fact that the eyeball does not have to be entirely removed induces some people to permit evisceration where enucleation would be refused. The objections commonly urged against it are the great reaction, which, as has been said, can be practically done away with if the simple operation is done, and the greater danger of sympathetic ophthalmia after it, an objection which, in my opinion, rests upon entirely insufficient ground. Another possible objection is that a small choroidal sarcoma whose existence was unsuspected might be eviscerated without being noticed, although retrobulbar metastases were already in progress. I know of one such case, and only one, where a subsequent evisceration of the stump showed a retrobulbar sarcoma. In the rare cases where there is the slightest question of anything of the kind this can generally be guarded against by careful inspection of the contents of the globe and the inner surfaces of the sclera. An infectious necrosis of the inner layers of the sclera has been mentioned as a possible complication of severe or long-continued panophthalmitis, which should contraindicate the use of the glass ball where the eye is eviscerated under these conditions.

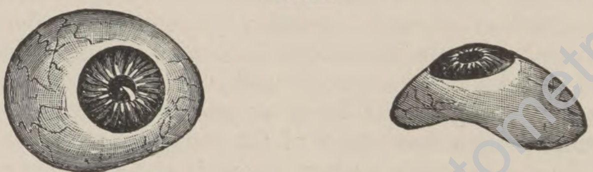
Opticociliary neurectomy is highly recommended by a few operators; but since it leaves the choroidal tissues intact as a breeding-ground for germs, which, it has been shown, can pass freely out of the optic nerve stump, it probably is the least efficient of any prophylactic operation for sympathetic ophthalmia. It should be recommended as a prophylactic only where one of the other operations is refused, or where, in the mind of the operator, the risk of sympathetic ophthalmia is so slight that he feels warranted in assuming part of the responsibility for it. In cases of absolute and painful glaucoma, it is often efficient, though not so certain in its results as evisceration. Its advantages are, that it will sometimes be consented to where other operations are refused, and that, although the eye thus saved is seldom as good-looking as an artificial eye, it is less troublesome, and in children it permits normal development of the orbit and temporal region. The evisceration can be performed later on in life

if it is desired for cosmetic purposes. The operation is more difficult and probably more dangerous than either evisceration or enucleation.

Artificial Eyes. An artificial shell may be inserted either into the cavity left by enucleation or over the stump obtained by an evisceration or one of its modifications. This should not be done in any event until the wound is entirely healed, which is usually in from ten days to three weeks after the operation. The use of an artificial eye should always be advised, for, in addition to its cosmetic value, it prevents the irritation of the conjunctiva which results when the lower lid becomes inverted. To insert an artificial eye, the upper lid should be drawn forward and the larger end of the shell, which should be moistened, slipped vertically under it. The lower lid should then be depressed, and the shell slowly rotated into its horizontal position. The shell is removed by slipping a small hook under the lower edge, and then making gentle traction upon the lower lid downward and while the shell is drawn forward. The "reform" eye has broad, smooth edges and is better adapted to some stumps.

As the enamel covering the eye soon loses its polish, great care should be taken to preserve it as long as possible. For this purpose,

FIG. 222.



Artificial human eyes.

as well as for the opportunity offered to flush the socket with some mild antiseptic lotion, the eye should always be removed during sleep, and, after careful washing, thoroughly dried. If, as sometimes happens, the lids adhere to the shell, a little vaseline may be introduced into the socket; but if the conjunctiva becomes roughened and catarrhal, it may be necessary to discontinue wearing the eye for a time, until the mucous membrane has received proper treatment.

As a rule, an artificial eye requires repolishing after eighteen months of continuous use.

It sometimes happens that an artificial eye cannot be retained on account of a deformity in the contour of the socket. This happens after burns and long-standing diseases of the conjunctiva which occasion cicatrices, and not rarely after the use of ill-fitting or badly polished artificial eyes. Under such circumstances an operation is necessary. One of the best of these is the procedure of Harlan.[1]

Transplantation into the orbit, after excision of the cicatrices, of Thiersch grafts or mucous membrane from the lips or vagina, has also been tried with success.

[1] de Schweinitz and Randall. American Text-book of Dieases of the Eye, p. 600.

CHAPTER IX.

DISEASES OF THE RETINA, OPTIC NERVE, AND ITS CEREBRAL ORIGIN.

By T. HOLMES SPICER, F.R.C.S.

THE RETINA.

The living retina is a transparent membrane of a slightly purple color, containing the expanded termination of the optic nerve. It is in contact with the choroid on its outer surface, and with the hyaloid membrane of the vitreous within. It extends forward as far as the ora serrata, where it ends in a wavy edge near the base of the ciliary processes. Beyond the ora serrata it is continued in a rudimentary form over the ciliary processes (pars ciliaris retinæ) and the back of the iris to the edge of the pupil (pars iridica retinæ). The parts of the retina which can be identified are the optic disk or papilla, at which the retina starts, and the yellow spot or macula lutea, a horizontal yellow oval patch at about two and one-half optic disk diameters outside the disk. At the centre of the macula is the fovea centralis, a small pit or depression, where all the layers of the retina, except that of the rods and cones, are absent; cones are present only in the fovea. The rods become numerous, and the cones decrease in number as they recede from the yellow spot.

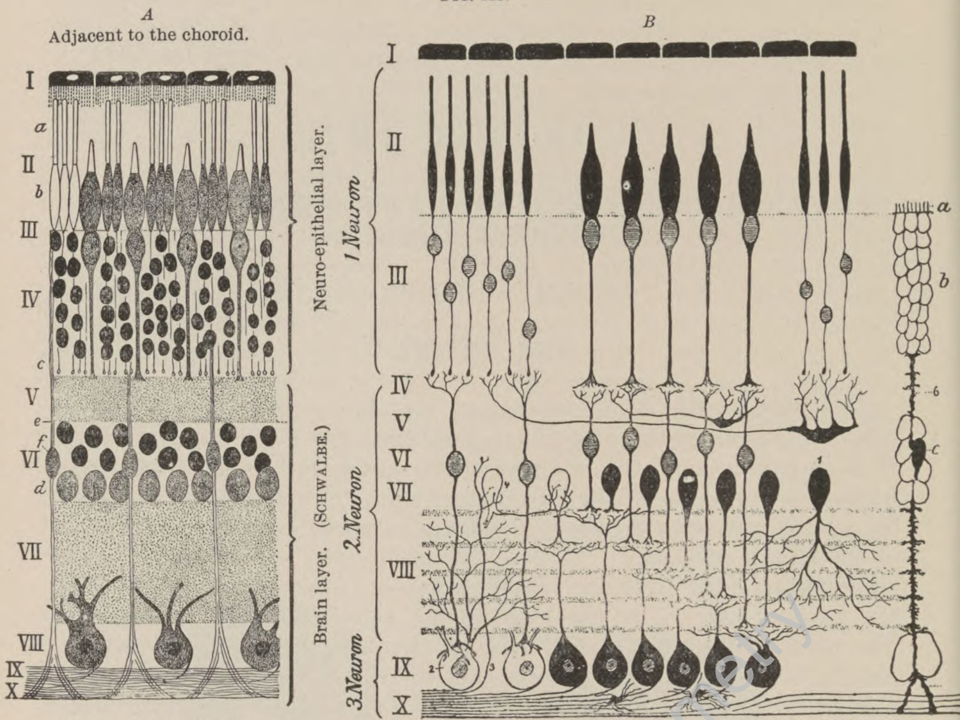
The fibres of the optic nerve within the eye, consisting of axis cylinders only, radiate in all directions from the disk and form the innermost or nerve-fibre layer of the retina. The retina, in sections made perpendicularly to its surface, is found to contain the following layers:

1. Nerve-fibre layer.
2. Ganglionic cell layer.
3. Inner molecular layer.
4. Inner nuclear layer.
5. Outer molecular layer.
6. Outer nuclear layer.
7. Rods and cones.
8. Hexagonal pigment cells.

There are also an inner and an outer limiting membrane, the latter lying between the outer nuclear and the rod and cone layer.

Most of the fibres of the nerve layer end in the cells of the ganglionic layer, but a few are continued into the inner molecular and inner nuclear layers. The molecular and nuclear layers of the retina con-

FIG. 223.



Adjacent to the vitreous.

Scheme of the structure of the human retina.

A. Horizontal section, hæmatoxylin stain. I. Pigment epithelial layer. II. Layer of rods and cones: *a*. External; *b*. Internal elements. III. External limiting membrane. IV. External molecular layer: *c*. Fibre layer. V. External granular layer. VI. Internal molecular layer: *d*. Spongioblasts; *e*. Supporting fibres of Müller; *f*. Nuclei of the same. VII. Internal granular layer. VIII. Layer of ganglion cells. IX. Nerve-fibre layer. X. Internal limiting membrane.

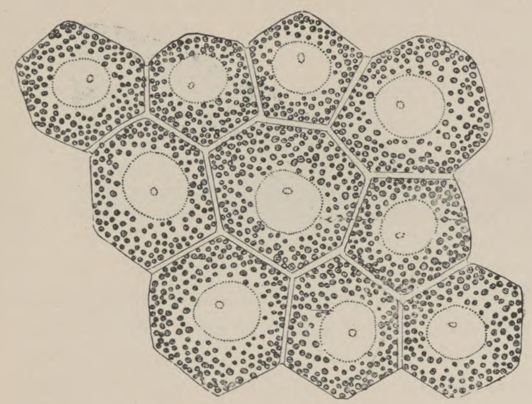
B. Demonstration after the method of Golgi. I. Pigment epithelial layer. II. Layer of rods and cones. III. Molecular and visual cells. IV. External plexiform layer. V. Layer of horizontal cells. VI. Layer of bipolar cells. VII. Layer of amacrine cells. VIII. Internal plexiform layer (fibre layers). IX. Layer of ganglion cells. X. Nerve-fibre layer: 1. Diffuse amacrine cells; 2. Diffuse ganglion cells; 3. Centrifugal nerve fibres; 4. Association-amacrine cells; 5. Neuroglia cells; 6. Supporting fibres of Müller.

sist of nerve cells or of their processes. The rods and cones are imbedded at their outer ends in the retinal pigment, a layer of hexagonal cells; the inner surfaces of these cells are prolonged into fine processes, which pass between and among the outer parts of the rods and cones. Under the influence of light the pigment comes forward into the anterior part of the cell and is found between the rods; in darkness it is collected in the body of the cell. The function of this pigment is to renew the visual purple or rhodopsin, after the latter has become bleached by the influence of light. There are certain other sustentacular or supporting fibres in the retina, passing from the anterior to the posterior limiting membrane—fibres of Müller.

The vessels of the retina are derived from the central artery and vein of the retina. These start at the disk, dividing and subdividing,

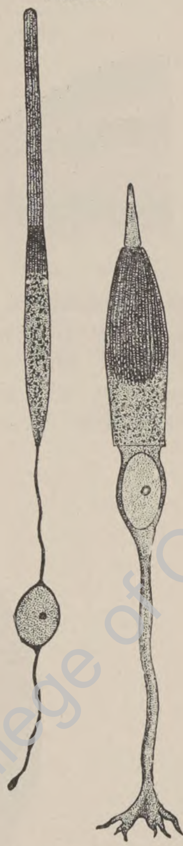
until they reach the periphery, but the branches do not anastomose with one another; the circulation is terminal. The importance of this is shown in the interference with the circulation of the eye by blockage of a vessel. No assistance can be obtained from the circulation in the adjacent parts. There is a limited collateral circulation between the retinal vessels at the margin of the optic disk

FIG. 224.



Pigment epithelium of the human retina.

FIG. 225.



Human rod and cone.
(GRAEFE-SAEMISCH.)

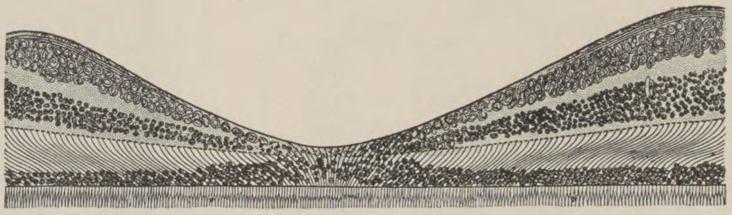
and branches of the short ciliary arteries, known as the circle of Zinn, and sometimes a retinal vessel may arise entirely from this source; it is then known as a *cilio-retinal vessel.* The vessels lie in the innermost or nerve-fibre layer; hence the outermost parts do not receive nutrition from the retinal vessels, but from the rich choroidal capillaries, with which they are in close contact. The retinal capillaries are extremely fine, and their meshes are closer at the yellow spot and its immediate neighborhood than toward the periphery; but at the fovea or central depression the capillaries are entirely absent.

The retina is the essential organ of vision: light entering the eye traverses all the layers of the retina until it reaches the posterior surface of the layer of rods and cones. The light stimuli are received by the rods and cones, and are transferred by means of the optic nerve to the brain, where they give rise to the impression of sight. The region of acute sight is at the fovea, a small depression at the centre of the macula which corresponds with an area at the centre of the visual field, one and one-half degrees in diameter. The elements of the retina at this point are 3 μ apart; this is expressed best by saying that two bodies in the visual field are not seen clearly

unless subtending an angle of at least 60″. The nerve supply of the fovea is more abundant than that of any part of the retina. In a case in which one-fortieth only of the field of vision was lost, Bunge found an atrophy of about one-quarter of the whole optic nerve.

The medullary sheath of the optic nerve fibres ends at the lamina cribrosa, but it is in rare cases present in the disk and extends to a

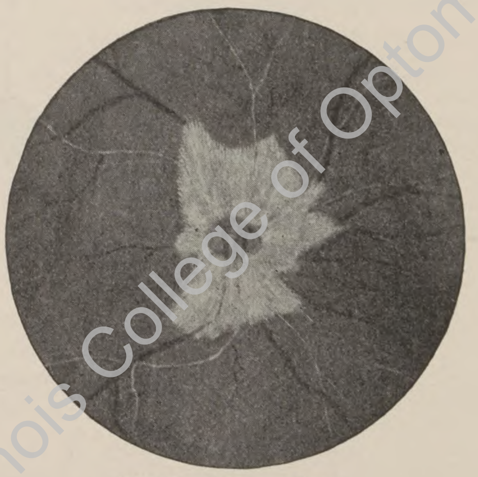
Fig. 226.



Section through the macula. (Graefe and Saemisch.)

varying extent into the retina. This condition is known as *opaque nerve fibres* or *retained nerve sheath.* (Fig. 227.) It may affect a portion or the whole of the disk, and may extend a long way into the retina. In rare cases separate islands of opaque nerve fibres are seen in the retina. These fibres, of a pure white or greenish-white color,

Fig. 227.



Opaque optic nerve fibres.

are densely opaque, with a striated surface and an edge which can be seen to spread out along the fibres of the nerve. The retinal vessels are seen on the surface of the opaque fibres or lie buried or partly buried beneath the surface. The portion of the retina affected with opaque nerve fibres is blind. After severe optic neuritis or neuro-retinitis, these fibres have been found to disappear.

Retinitis.

Inflammation of the retina may occur as the result of an injury or from the concentration of brilliant light of the sun or of the electric arc upon it; but, as a rule, it is the manifestation, often the only one, of grave constitutional disease, such as albuminuria, general arterial sclerosis, syphilis, leukæmia, or diabetes.

Hyperæmia or congestion of the retinal capillaries cannot be recognized, the retinal capillaries being themselves invisible, but the condition of the visible bloodvessels may often enable one to diagnose a condition of general retinal hyperæmia, such as largeness or tortuosity of the arteries, and distention, tortuosity, and dark color of the veins.

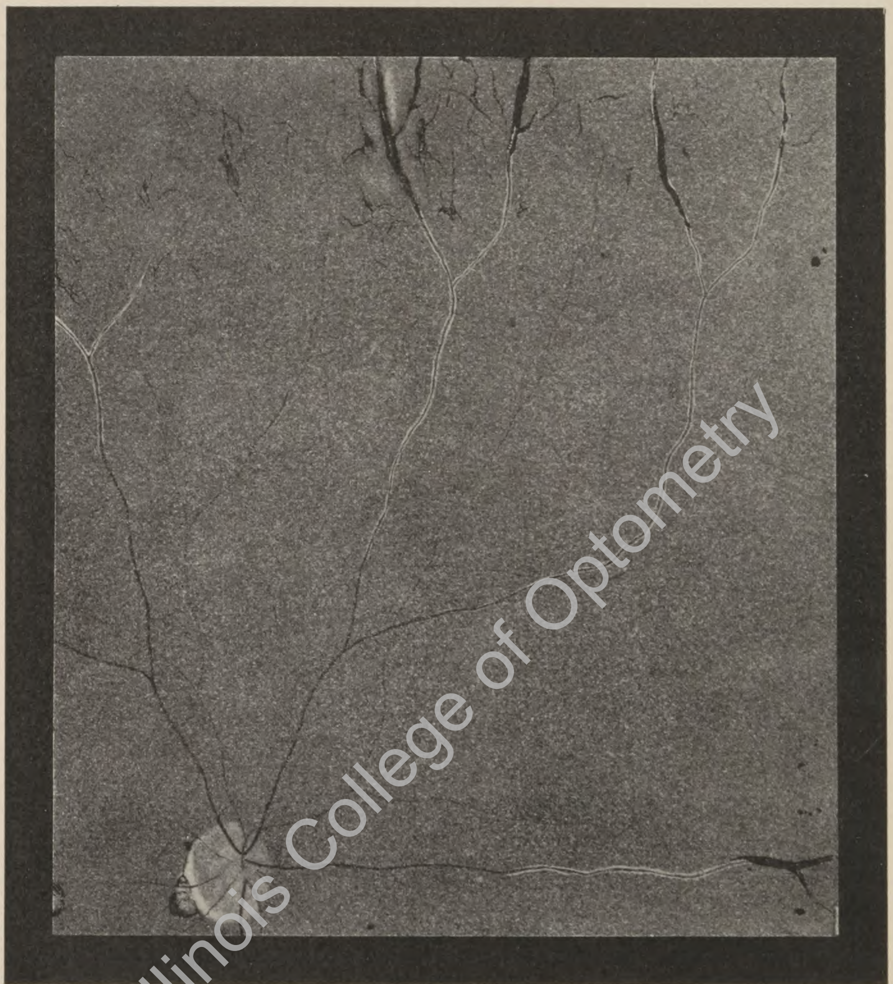
All pathological changes in the retina show themselves by a loss of transparency, the affected parts become gray or white and opaque, the vessels are indistinct or entirely obscured, and the underlying choroid less clearly seen than in other places. In some cases the red reflex from the choroid is dulled, so that the retina appears to have a smoky hue. This opacity may be diffuse and occupy a large part of the retina, or it may be limited to one region, or may show itself in circumscribed areas separated by healthy retina.

Inflammatory changes are often accompanied by hemorrhages into the retina. These may be linear in shape, and may bear a relation to a visible bloodvessel; they may be punctate, streaked, or flame-shaped, owing to the direction of the nerve fibres in which they lie. They are generally rounded in the deeper parts of the retina; the only symptoms present, as a rule, are diminution of sight with occasional flashes of light or flickerings, distortion of objects, such as straight lines (metamorphopsia), diminution in size of objects (micropsia), or night blindness. In some cases floating specks are seen before the eyes, which are ascribed to biliousness; and ophthalmoscopic examination may be the first indication afforded of severe constitutional disease.

Syphilitic Retinitis. Syphilitic retinitis may show itself either in association with choroiditis, as choroido-retinitis, or as a pure retinitis. It occurs during the secondary period of syphilis, between the sixth and the eighteenth month after the primary sore. It occurs in congenital, as well as in the acquired disease, and generally attacks both eyes. It runs a very chronic course, lasts many months, and shows a marked tendency to recur. The ophthalmoscopic signs are an exudation into the vitreous, generally into its posterior part. This exudation is very fine, but can generally be resolved into actual dust opacities by the ophthalmoscope; it obscures the fundus and may hide the deeper parts entirely from view, but it is often possible to recognize through the haze the hyperæmic condition of the optic disk, with great enlargement and tortuosity of both arteries and veins. There may be spots or white areas of exudation into the retina, and hemorrhages are often present. In the

later stages the vitreous clears; the arteries are then found diminished in size, and both arteries and veins are sheathed in white. The retina remains opaque and fibrous looking, and contains pigment along the sheaths of the vessels or scattered irregularly, chiefly at the periphery. (Fig. 228.)

FIG. 228.

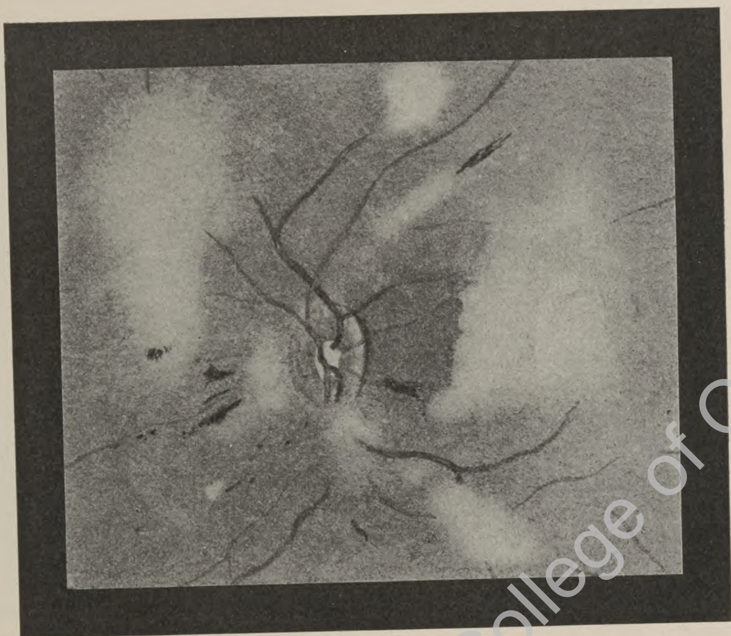


Atrophy of retina. Pigmentation of sheaths of retinal vessels after syphilitic retinitis. Note white lines along the retinal veins; pigmentation of veins at periphery, arrangement of pigment between the main vessels resembling retinitis pigmentosa.

The loss of sight bears no relation to the ophthalmoscopic changes. It may from the first be much reduced, and, after subsidence of the disease, may remain so; while in other cases the sight generally may

be considerably restored, but blind areas (scotomata), or a ring-shaped area of blindness (annular scotoma) may be left behind. Treatment should be begun without delay. If mercury be given to the limit of safety, the course of the disease may be shortened and some of its worst effects avoided. Inunction is perhaps the most satisfactory method of giving mercury; it should be pushed until there is slight tenderness in the gums. This may be alternated with subcutaneous injections of mercury, or with mercury with chalk, taken by the mouth. The mercurial treatment should be carried out until the disease has come to an end and sufficient time has passed to

FIG. 229.



Albuminuric retinitis in a case of acute nephritis, showing areas of soft-edged, œdematous-looking exudation into the retina, with hemorrhages.

render recurrences improbable. To this treatment should be added Turkish baths, subcutaneous injections of pilocarpine, and in the later stages iodide of potassium. At the same time dark glasses should be worn and all use of the eyes prohibited; the glasses should be domed, of neutral color, and rather dark shade, with sides protected by gauze or crape, to exclude light; some surgeons speak highly of spectrum blue glasses. It is doubtful whether other local measures, such as leeching or counter-irritation, have any effect.

Albuminuric Retinitis. Inflammation of the retina associated with renal disease occurs very often during the course of a chronic

interstitial nephritis or granular kidney. It occurs also in chronic parenchymatous nephritis, in the kidney disease of pregnancy, and also less commonly during an attack of acute nephritis. (Fig. 229.) It is convenient to class all these varieties under the heading of albuminuric retinitis, although it must be understood that albumin is not present constantly in all cases; it occurs in two forms, at least: the inflammatory and the degenerative.

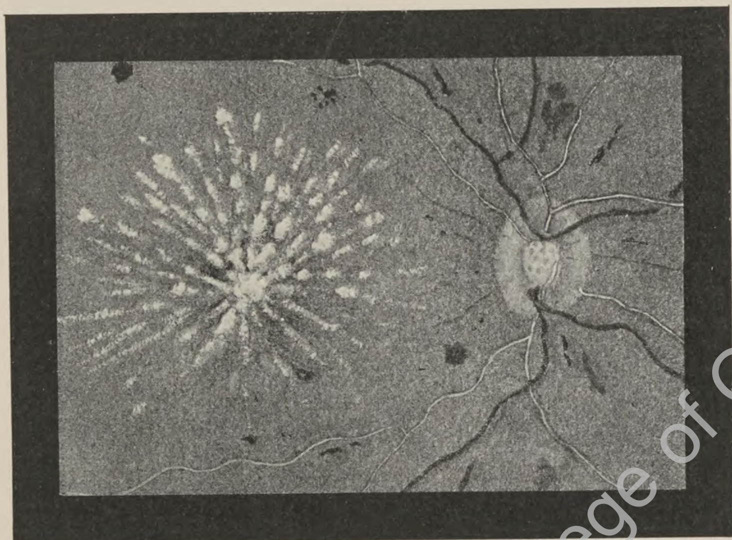
Inflammatorg Retinitis. Where the disease is running an acute course, whatever be the fundamental nature of the kidney affection, we meet in the retina with soft white flocculent patches of exudation, combined with œdema covering large areas, with swelling and haze of the disk. Hemorrhages are sometimes present as small red points or flame-shaped masses of blood in the nerve-fibre layer. This form of retinitis is not always associated with much impairment of vision, and is seen less frequently than the other form. It is most commonly met with in the chronic large white kidney stage of nephritis; it persists for a few weeks, and, with general treatment, it may disappear and leave no trace. This exudative or inflammatory form of retinitis is frequently accompanied by a great deal of exudation into the optic nerve, producing a condition closely resembling the optic neuritis of intracranial disease.

The other form of retinitis, the *degenerative*, is sometimes seen after subsidence of the acute exudation, but generally occurs independently. It consists of very brilliant dazzling white spots about the macular region. Its most characteristic form is very like an asterisk radiating from the yellow spot. Each dot of which the asterisk is made up has a sharply defined or hard edge, and the surrounding retina appears to be darkened, possibly from contrast with the brilliant exudation. The exudation consists mainly of granules mixed with fatty deposit in the nervous and supporting elements of the retina, and probably owes its peculiar arrangement to the folds into which the retina is thrown by œdema. Hemorrhages are generally present also, and may be punctate, striated, linear, or flame-shaped. The tendency is for the exudation to become absorbed and for sight to be somewhat improved. It is rarely entirely absorbed, however, and months after a few dots may generally be seen near the yellow spot; the hemorrhages also become absorbed slowly. A peculiarity which is seen in some cases is pigmentation of the retina, which has been found on microscopic examination to lie outside the external limiting membrane. In severe cases of albuminuric retinitis accompanied by marked papillitis the recovery of sight never proceeds very far, and if the disk becomes atrophic, vision may be almost entirely lost. Sight may be lost also in kidney disease without the occurrence of retinitis. The sight fails rapidly and completely without any cause being discernible by the ophthalmoscope; but after a few hours recovers slowly. The patient has headache, vomiting, and the other symptoms of uræmia, and the blindness is *uræmic amaurosis*. In the albuminuria of pregnancy the retinitis may come on comparatively

early, or may be delayed until near the end of the pregnancy. It follows an acute course, and is attended by great disturbance of function, but complete recovery is often obtained.

Recovery is more likely to occur in the inflammatory or exudative form of retinitis than in the degenerative form. Changes in the vessels are marked in the degenerative form. The small arteries are thickened and rigid, especially the inner coat, and their lumen becomes diminished; the capillaries participate in this rigidity. (Fig. 230.) This change shows itself very clearly by the ophthalmoscope, as has been described by Marcus Gunn. The smaller arteries of the retina have their central light streak wider and more brilliant than usual,

Fig. 230.



Albuminuric retinitis. Granular kidney. Note hard-edged "asterisk" exudation at *y s*, the silver-wire condition of the arteries, and the punctate and linear hemorrhages.

so that the whole vessel appears like a piece of silver, or rather, of gold wire, and gives one the impression of being hard, round, and tense. At the same time the artery shows signs of degeneration in the form of small bright spots in its coat. Where it crosses the veins the blood current in the latter is interfered with, so that the column of blood appears to be cut in two, and the distal part is distended by the obstruction. In more advanced arterial disease there are slight inequalities in calibre of the arteries in different places, and, occasionally, small aneurisms may form on them. The distended veins sometimes rupture, owing to degeneration of their coats from stasis of the blood within them, and they may undergo fusiform enlargement. Hemorrhages may occur also from the arteries them-

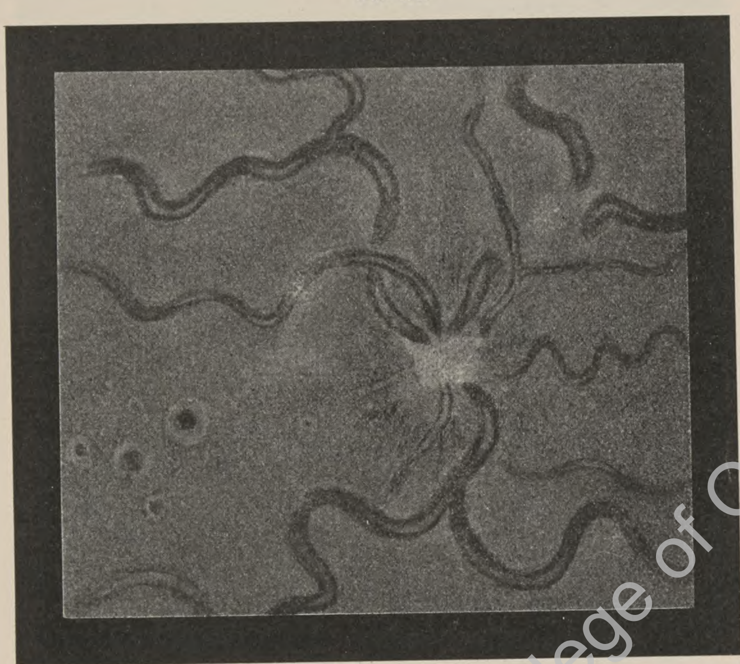
selves. One of the common results of this form of arterial degeneration is the occurrence of an area of thrombosis of the vein at the point where it is crossed by an artery. This is followed by an inflammatory exudation, completely obscuring the vessels at the place of contact, while hemorrhages are poured out from the vein beyond the obstruction. These changes in the vessels are seen generally after about forty years of age, but may appear earlier—indeed, almost at any age. They may be said to be almost characteristic of granular kidney, although they are seen frequently in patients in whom no other signs of granular kidney can be found, except, perhaps, a hard, incompressible artery at the wrist.

Prognosis. This is to be regarded from the point of view of recovery of vision, and also with respect to the duration of life. As has been said, the exudative or inflammatory form of retinitis may be absorbed entirely and leave the vision little impaired. The degenerative form takes much longer to become absorbed, is less likely to be absorbed at all, and may leave permanent changes in the macular region, which may interfere greatly with vision. The pregnancy forms are likely to recover, provided that pregnancy is near its end, or if it can be determined by premature delivery. As regards the duration of life in the exudative forms accompanying parenchymatous nephritis, although the retinal changes may be very great, the prognosis is not extremely grave, for the condition of the kidney may be recovered from. In the degenerative forms accompanying granular kidney the duration of life is short; in hospital patients the average duration of life has been noted by Miley among 45 cases to be under four months, and the extreme duration under two years; but other observers have noted less unfavorable results. The prolongation of life seems to depend upon the amount of care that can be taken of the health. Thus another observer found among hospital patients that all the men died within two years, and 68 per cent. of the women, and among private patients only 59 per cent. of the men died, and 53 per cent. of the women. A few exceptional cases have been recorded in which life was prolonged for seven or even twelve years.

Diabetic Retinitis. Although the existence of an inflammation of the retina peculiar to diabetes is not admitted by many authors, yet retinal changes are met with in diabetes which are distinct from those met with in any other disease, and sufficiently like each other to justify their recognition as a separate variety. The form which diabetic retinitis takes is that of a group of brilliantly reflecting dots or areas of degeneration in the retina, varying much in size and grouped around the yellow spot. The appearance of each degenerated area is much like that of the individual spots met with in albuminuric retinitis, but their arrangement is not like the spokes of a wheel, radiating from the yellow spot, but like the circumference of a wheel arranged around it. At the same time there are numerous punctate and linear hemorrhages in the retina. This form of exudation persists for a very long time.

Leukæmic Retinitis. Owing to the poverty of the blood in coloring-matter, one of the most striking features of the fundus in this disease is that the color of the choroidal reflex, instead of being a full, rich red, is of a light-yellow color. The retinal veins are large, flabby, and tortuous, and have the appearance of flattened bands. Retinal hemorrhages are present with white masses of exudation, due to extravasations of white blood cells into the retina. These spots are sometimes surrounded by a fringe of colored blood. (Fig. 231.)

FIG. 231.

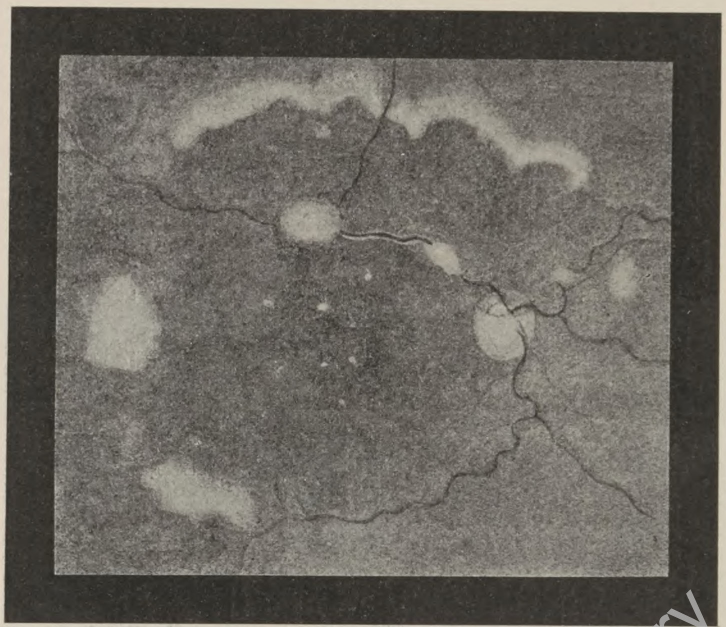


Leukæmic retinitis. Note the indistinctness of the disk; the enormously distended veins; and the hemorrhages at the yellow spot surrounded by a light halo.

Treatment. The treatment of albuminuric, diabetic, and leukæmic retinitis is the treatment of the disease which is the cause of the retinitis, and calls for no remark here, except that rest of the eyes should be enjoined and the use of dark protective glasses. In retinitis occurring during pregnancy, the question of inducing premature labor often arises. If the retinitis be severe, it is advisable to induce labor; but this should be postponed as long as possible, if it can be done with safety, in order to save the child.

Hemorrhagic Retinitis (Thrombotic Retinitis). The ophthalmoscopic appearances in this disease are the formation of a very large number of small flame-shaped hemorrhages all over the retina or

Fig. 232.



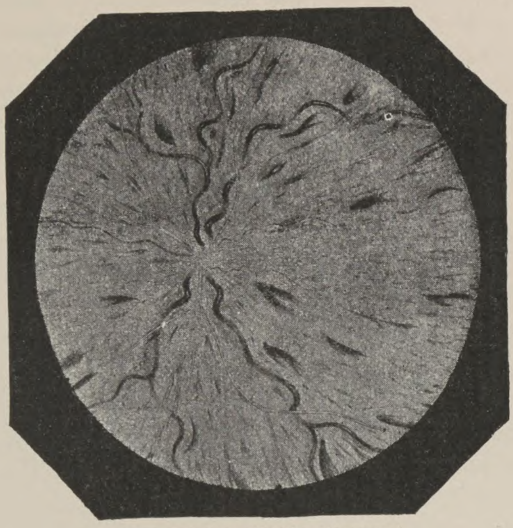
Hemorrhagic retinitis. Venous thrombosis: upper temporal vein, showing two patches of thrombosis; upper nasal vein, showing one patch; white exudation concentric with optic disk. (General view of inverted image.)

Fig. 233.

Venous thrombosis. Upper venous vein, showing patches of thrombosis. White exudation concentric with O. D.; portion of preceding figure seen by the erect image; the thickening of the sheath of the vein should be noted.

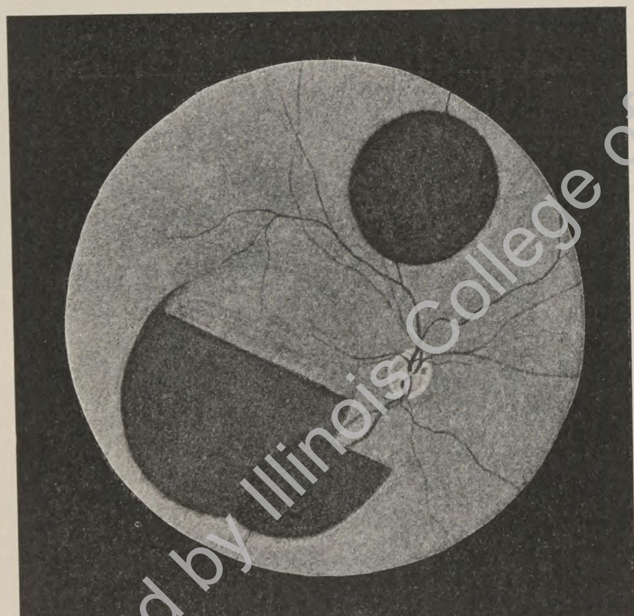
over a portion of the retina drained by a single vein. (Figs. 232 and 234.) At the same time the veins in this region are enormously dis-

FIG. 234.



Hemorrhagic retinitis. (JAEGER.)

FIG. 235.



Note subhyaloid hemorrhage at yellow spot, which has burst through its anterior limiting membrane and formed a circumscribed hemorrhage; a second subhyaloid hemorrhage is seen at the upper part of the fundus.

tended, tortuous, and dark in color, while the affected part of the retina is generally œdematous, and may contain spots or areas of exudation. The cause of this affection is thrombosis of the trunk of the vena centralis retinæ, or of a branch of it; it usually occurs in one eye only. It is caused by general vascular degeneration, with disease of the valves of the heart or of the arteries. It is said to occur among the gouty; it certainly occurs among those who have no discoverable disease of any kind. It is sometimes secondary to orbital disease, such as cellulitis, erysipelas, or disease of the cavernous sinus, and the thrombosis here probably travels along the course of the vein until it reaches the eye; in such cases the optic nerve is generally left atrophic. Hemorrhagic retinitis sometimes ends in hemorrhagic glaucoma.

Subhyaloid Hemorrhage. This consists in the formation of a large circular hemorrhage on the surface of the retina beneath the hyaloid membrane, and, according to Fisher's observations, beneath the membrana limitans interna, from the rupture of a vessel, probably a retinal vein. It is generally confined to one eye, is single, and occupies the yellow spot region, but it may, in rare cases, be associated with hemorrhages in other parts. Sight is at first much impaired, but as the hemorrhage gets slowly absorbed the retina again becomes exposed, and vision is restored, or the blood may suddenly be diffused into the vitreous through a rupture in the hyaloid membrane. (Fig. 235.)

Hemorrhages are also met with in pernicious anæmia, malaria, amyloid disease, purpura, and burns of the skin. Large retinal hemorrhages which sometimes burst into the vitreous are seen occasionally in young adults without apparent cause. Some of the patients are anæmic, others are apparently in perfect health; such patients are generally subject to constipation and epistaxis, according to Eales, who first described them. The treatment of all these forms of retinitis should be carried out on general principles. Small regular doses of blue pill, followed, if necessary, by a saline, are advisable; it is also of advantage to give iodide of potassium.

Pyæmic Retinitis. This results from infection of the eye from a septic centre elsewhere in the body, as in ulcerative endocarditis. It may be produced also by a decomposing retained placenta or by any other decomposing material in the uterus. It shows itself in the earliest stages by hemorrhages and white patches in the retina. It results in panophthalmitis, which may be double and lead to total loss of both eyes.

Retinitis Proliferans and Retinitis Striata. In this disease dense masses of fibrous tissue project forward into the vitreous from the retina. These tufts of fibrous tissue contain bloodvessels of new formation; the delicate new bloodvessels give way, forming periodically fresh hemorrhages. The fibrous tissue probably has its origin in the organization of the blood that is poured out; small but less extensive growths of vessels may occur in syphilitic retinitis. A closely allied form is that to which the name of *retinitis striata* has been given, in

which the bands of fibrous tissue form in the retina itself, and generally follow the course of the bloodvessels.

Retinitis Circinata. This is a rare affection, in which large areas of brilliant white exudation with dots of dark color on them are formed in the region around the yellow spot. The central portion of the retina itself has undergone much degeneration, and is gray and opaque; the appearance of the white patches in the retina is rather like that of passover bread. Hemorrhages generally accompany the disease. It occurs mainly in very old people. (Fig. 236.)

FIG. 236.

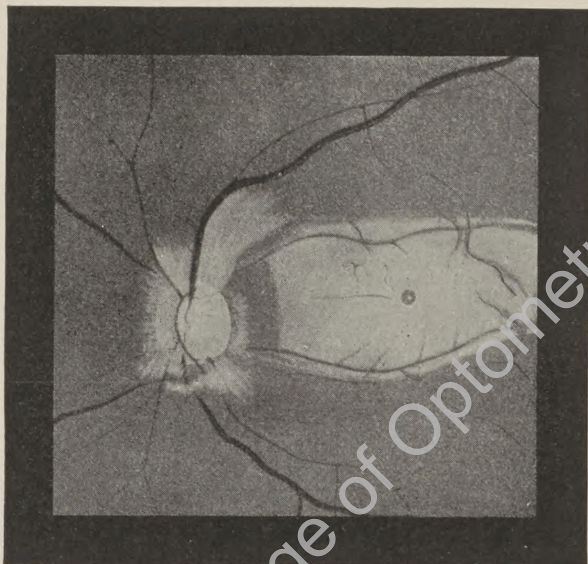
Retinitis circinata. Note the gray degeneration of the retina at the yellow spot, and the white exudation concentric with the yellow spot, having the appearance of passover bread.

Symmetrical Disease of the Macula in Young Children. This affection, first described by Tay, associated with disease of the cerebral cortex, is a rare disease; it occurs during the first two years of life, generally among the offspring of Jewish parents. A white patch of exudation having a cherry-red spot in the centre is seen at the macula; the optic nerve slowly atrophies, the child becomes blind, and degenerates mentally until death ensues after some months.

Embolism of the Central Artery of the Retina. When this occurs, there is sudden and complete failure of sight. The patient is about his ordinary occupation and is conscious of something peculiar in

his sight. On covering one eye he finds that there is only faint perception of light in the affected eye. (Fig. 237.) When examined by the ophthalmoscope after a few hours the arteries are found to be much shrunken, but generally not quite bloodless; the veins are of normal size or rather smaller, but tend to increase in size away from the disk. The whole retina is whiter than normal, the whiteness being most marked around the yellow spot. The fovea appears by contrast brightly red, as if there had been a hemorrhage in it. This appearance is known as "the cherry-red spot at the macula." The whiteness of the retina is due to œdema, and this is most marked just around the yellow spot, where the retina is thickest; the cherry-

FIG. 237.



Embolism of central artery of the retina. (LIEBREICH.)

red spot at the fovea is owing to the red of the choroid being seen through the retina, which is very thin at this spot. The cherry-red spot has been seen within twenty minutes after embolism has taken place. The column of blood present in the vessels is sometimes broken up and moves about in an irregular manner, sometimes from one vein to another; sometimes in a reverse direction to the normal. All the small vessels in the region of the macula stand out very clearly against the opaque white retina. After the first few days there is frequently slight improvement in sight, owing to partial restoration of the circulation from anastomosis of small vessels situated around the entrance to the optic nerve, but the improvement is very slight; the œdema clears up in a few weeks, and atrophy of the disk follows.

Sometimes only a branch of the central artery is affected, with the corresponding portion of the retina. In one published case the macular region was supplied by a cilioretinal artery, and central vision was retained, although all the other parts of the retina were blind. A case has been seen recently in which embolism of one artery was followed by a similar accident in the other eye.

Cause. The most commonly assigned cause is the separation of an embolon from a diseased valve of the heart. Other causes are atheroma of the aorta or other large vessel, aneurism, pregnancy, or Bright's disease. But cases are not rare in which there is no cause of this sort to be found by most careful examination, and it is probable that many of the cases presenting typical features of embolism are really cases of sudden thrombosis of the central artery, due to endarteritis.

Treatment. As to treatment, nothing can be said to be beneficial with certainty. Paracentesis of the anterior chamber and iridectomy have been tried without result; massage of the eye may be tried with the hope of causing the embolon to move to some more distant part of the circulation; it has been successful, but it must be applied early and with force.

Thrombosis of the Central Retinal Artery. This gives rise to symptoms and ophthalmoscopic appearances identical with those of embolism, the main difference being that in thrombosis the patient is warned by temporary failures of sight which pass away, until one occurs which does not clear up.

Quinine Blindness. (See Optic Nerve.)

Effect of Light on the Retina. The effect of light on the eye shows itself in the conjunctiva and in the retina. Exposure to the sun, producing sun blindness, or to the electric arc, as a rule, causes intense conjunctivitis, a condition analogous to blistering of the skin by direct sunlight. Thoughtless exposure of the eyes to the sun, or to the rays of a powerful arc lamp, may produce results on the retina which are sometimes permanent. Many instances are recorded in which patients have stared at the sun during an eclipse. The result has been in some cases a persistent positive scotoma, producing indistinctness or a blur over every object directly looked at. In other cases, without the sight being at all dim, the consciousness remains of a colored spot in the centre of the field, seen generally when a white background is looked at. Other cases have been recorded in which a permanent central scotoma with loss of acuteness of vision has been left behind. Observation of the fovea in such cases has shown it to be swollen, or to have a hemorrhage at its centre, or, at a later stage, to be atrophic. Treatment should be by rest and dark glasses worn for a prolonged period.

Atrophy of the Retina. Atrophy of the retina may occur as the result of long-continued previous inflammation. It may be the consequence of an embolism or thrombosis of the central retinal artery.

Or it may be a special affection characterized by the formation of new pigment, and known as retinitis pigmentosa.

Retinitis Pigmentosa. This disease is characterized at its onset by loss of power to see at night or in twilight (night blindness[1]), the vision remaining good during daylight. If the field of vision be taken at this time, it will be found normal or nearly so in bright light; but if the illumination be diminished, some reduction in the size of the fields will be found. As the disease progresses the field becomes contracted, even in bright daylight, and the contraction may advance so far that the patient has difficulty in finding his way about; in extreme cases the field is reduced to a mere point. Even in this stage central vision may be almost unimpaired; in some cases the whole of the field is not lost, but a zone or belt of the retina becomes blind, giving rise to a ring scotoma. By ophthalmoscopic examination the retina is found to contain a large amount of pigment in its anterior layers, deposited in the form of dots or islands, shaped much like bone corpuscles, having branches which communicate with other neighboring dots. Pigment is deposited also along the sheaths of the smaller vessels. In more advanced cases the red background of the eye appears to be covered with a delicate black lacework; in its most severe form the pigment is so dense that little of the red choroid is visible. The general arrangement of the pigment is in the form of a zone situated about midway between the periphery of the retina and the optic nerve. This belt is densest at the centre, and thins off at its inner edge, toward the disk and also toward the periphery. The retinal vessels become reduced in size; the arteries may be mere threads; the disk undergoes a peculiar dirty yellow atrophy known as *post-retinitic* or *waxy atrophy;* the lens may become affected with posterior polar cataract, and opacities may appear in the vitreous. Although this is the usual character of the disease, cases are occasionally met with in which night blindness and loss of fields are present and some post-retinitic atrophy of the disk is seen, but in which no pigment can be seen in the retina, or in which, instead of pigment, a number of soft-edged rounded yellow-white spots are seen. These two conditions are spoken of as *retinitis pigmentosa without pigment* and *retinitis punctata albescens.* The retinal hexagonal pigment is gradually absorbed or travels forward to the anterior layers of the retina, so that the choroidal vessels appear to stand out very clearly. There is some doubt at present as to whether this disease should be considered as choroidal or retinal in origin, as, according to Wagenmann, if the choroidal circulation is interfered with by division of the ciliary vessels, a migration of pigment forward into the retina takes place like that seen in retinitis pigmentosa. The disease is first met with in

[1] In consequence of the confusion which has arisen with regard to the words nyctalopia and hemeralopia, they being used in opposite senses by English and Continental writers, it is better to use the terms night and day blindness, which explain themselves.

childhood, or about puberty, and advances slowly to almost complete blindness after middle life; it attacks both eyes. Its cause is unknown. It is frequently hereditary, and occurs in those whose parents were blood relations before marriage. Other defects of the nervous system are often present, such as deafness and want of mental power. No treatment is known to be successful in this disease. Iodide of potassium and strychnine may be tried, with the application of the constant current.

The prognosis is bad, although complete blindness may not come on until very late in life.

The atrophy which follows syphilitic retinitis, especially in childhood, is often similar in appearance and course to true retinitis pigmentosa; but, as a rule, there is some evidence of involvement of the choroid in the syphilitic affection.

FIG. 238.

Detachment of the retina. (JAEGER.)

Detachment of the Retina. The retina is continuous with the optic nerve at the disk, and is attached to the choroid at the ora serrata; but between these points it is held in apposition with the choroid only by the support or pressure of the vitreous within it. It is liable to be detached from its position by various causes, such as injury, extravasation of blood or serum, by traction from within, from bands in the vitreous, by tumors of the choroid, or cysticercus. It is met with most commonly in myopic eyes. (Fig. 238.)

The cause of the detachment has been accounted for in many ways—by exudation of fluid from the choroid, by sudden extravasation of blood from the choroid. Neither of these theories explains the large number of cases in which the detachment comes on suddenly

without sign of hemorrhage. It is owing to the work of Leber and Nordenson that the theory of shrinkage of the vitreous was established and most of the difficulties of the subject answered. According to their observations, the vitreous becomes fibrillary in structure while retaining its transparency. This change is due to a shrinkage from inflammatory processes in the choroid or ciliary body; serous fluid becomes poured out into the vitreous chamber to fill the vacuum caused by the shrinking. The traction on the retina produced by the shrinking vitreous leads to rupture of the retina. The serous fluid lying in the vitreous chamber passes through this rent into the subretinal space and allows the retina to become suddenly detached. More recently Raehlmann has explained the detachment on the *diffusion* theory: the fluid behind the retina is more albuminous than that in front of it; diffusion tends to take place more rapidly toward the fluid of greater density—that is, from the vitreous to the subretinal spaces—than in the opposite direction. Although this theory may explain some of the slow detachments, it hardly suffices for those of sudden onset.

The detachment may take place at any part. It is less common at the macular region than at the periphery; but wherever it begins, it soon settles to the lowest part of the retina, owing to gravitation of the fluid, while the part first detached may become reapplied. It may remain stationary, but it generally progresses until the whole retina is detached, so that in a *post-mortem* examination of the eye the retina appears as a cord going from the optic disk to the back of the lens, containing the shrunken remains of the vitreous, and spreading out thence to the ora serrata, forming an umbrella or convolvulus flower-like detachment. The evidence of inflammatory changes in the eye is generally present in the signs of iritis or iridocyclitis or opacities in the vitreous. Secondary cataractous changes in the lens generally appear late in the disease, with a reduction of tension, except in those cases where the detachment is caused by a choroidal tumor. Myopic eyes are those most subject to detachment of the retina, but it is not always those in which the amount of myopia is highest which suffer from detachment. Vision may not be much affected if the yellow spot be not involved, but there is always a considerable loss of field, which may be detected by the perimeter, by the hand, or by the light projection test; the part of the field which is lost will correspond with the opposite portion of the retina. If, owing to opacities in the media, it is not possible to use the ophthalmoscope, it is generally possible to diagnose the presence of a detachment by testing the projection of light. In the first stages of a detachment examination of the field of vision alone is insufficient, as the retina may retain its function for some time after the detachment where the latter is not very deep. The ophthalmoscope shows a changed color in the reflex from the fundus over the detached area. The best way to see this is to observe the fundus reflex from a distance of about 14″ with the ophthalmoscope mirror alone, and to get the patient to look

in various directions, so that the whole of the retina is brought under observation, and one part may be compared with another or with the other eye. The reflex, even in recent cases, is generally slightly duller over the detached area than elsewhere, and in old detachments the retina may appear opaque and gray. It may often be seen to float about with movements of the eye. The detached area should then be looked at by the direct method, its refraction estimated and compared with that of other parts. If one part of the retina is much more hypermetropic or less myopic than another, suspicion should be directed to detachment of the retina. The retinal vessels in the detached area appear much darker than normal, owing to loss of their central light streak and to the difference in transillumination. In their course toward the periphery they can be seen to disappear into folds and depressions in the retina; rents in the retina may be seen at times, showing the bright choroidal reflex behind. In some cases of shallow detachment the retina appears to be thrown into innumerable fine ripples which have very much the appearance of the vessels of the choroid seen through the retina; it is possible that this appearance may also be due to detachment of the choroid with the retina.

In determining the cause of the detachment, regard should be paid to its seat and extent, its shallowness or depth, its translucency and immobility, the condition of the vitreous, and the hardness or tension of the eye. New-growths of the choroid generally form globular prominent steep detachments, sometimes dark in color, owing to the pigment they contain, sometimes showing vessels not of retinal origin. The vitreous is not opaque, and the tension of the eye often is raised. If the detachment be due to shrinking of the vitreous, there will be vitreous opacities, a widespread detachment, a floating retina, probably containing rents through which the choroid may be seen.

Treatment. The treatment should be directed toward producing absorption of the exuded fluid. For this purpose the most efficacious agent is complete rest in bed; the patient should be kept on his back for a month or six weeks, his diet should be limited in regard to fluids, and free action of the skin should be provided, either by vapor baths, which should be given in bed, or by the subcutaneous administration of pilocarpine. This may be combined with the use of the iodides of ammonium and potassium internally. At the same time the eye should be kept bandaged under moderate pressure. If more rapid disappearance of the fluid be desired, the situation of the greatest collection of fluid should be made out by the ophthalmoscope, and the fluid tapped through the sclerotic. This is done best by a broad needle or a Graefe knife, which should be introduced through the sclerotic into the subretinal space in the equatorial region at a spot between the insertion of the muscles. If the knife then be turned upon its long axis, an opening will be made beside it, which will allow the albuminous fluid to run out of the eye. Before introducing the knife the conjunctiva should be displaced by the fixation forceps,

so that when the knife is withdrawn the conjunctiva may slip back and the wound in the sclerotic be covered. Fluid will go on draining away into the subconjunctival connective tissue after the knife is withdrawn. An addition to this plan, which has been recommended and has met with some success, is to burn the sclerotic slowly with a cautery through its outermost layers, until the choroid is just reached. By this means an adhesive inflammation is set up in the choroid, which aims at binding the retina to itself by the after-contracting process. No method of treatment is very hopeful. The retina may become reattached for a time, but it is frequently displaced again on the patient resuming ordinary routine. It is not possible to overcome the tendency to contraction in the vitreous, and, if the retina becomes reapplied, it is likely to be displaced again by continuance of the contracting process.

Other methods of treatment have had success for a time. Schoeler's method of injecting iodine into the vitreous cavity, which scored some successes in its author's hands, led to disastrous results in other cases. Deutschmann's method of making a puncture through the sclerotic, choroid, and retina into the vitreous, and cutting on each side of this track to divide the bands in the vitreous, has not turned out more successful than other methods of treatment.

Cysticercus of the Retina. This is a very rare disease. Its diagnosis depends mainly upon the appearance of the parasite. It is subretinal as a rule, it has the appearance of a flattened cyst, it is light gray in color, with light edges, and undergoes spontaneous movement; the head may occasionally be made out. The only treatment is to cut down upon it and remove the cyst.

Injuries of the Retina. Besides detachment, the retina sometimes after a blow on the eye will be found to have an injured area, white or nearly so in color, with ill-defined edges. This condition, which usually passes away in a few days, is probably due to a local traumatic œdema. It is known by the name *commotio retinæ*.

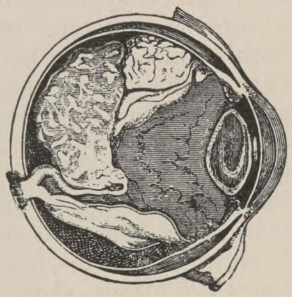
Holes at the Macula. After injuries to the eye, especially from concussion by a stone, ball, or other large object, in which the globe is not ruptured, there is frequently associated with loss of central vision a remarkable appearance at the yellow spot. The retina ceases abruptly, so that there appears to be a circular hole in it at the centre of the yellow spot, equal to about one-third the diameter of the disk. The floor of the hole is formed by the choroid, and is depressed a measurable distance behind the retina. Although no pathological examination of the condition has ever been made, it is highly probable that this appearance is really due to a hole at the fovea, caused by rupture of the retina by *contrecoup:* the elastic retina retracts and leaves a clear round hole whose edges are placed at a distance which can be readily appreciated from the choroid behind. There is usually considerable loss of vision.

Glioma of the Retina. This is the only form of tumor that attacks the retina. It occurs in early childhood, before the age of three

years. It is sometimes congenital, and is met with in rare cases at a later age. It starts from one of the granular layers of the retina, and either grows inward toward the vitreous or outward, producing detachment of the retina. It consists of cells arranged in long tubes around wide bloodvessels. The cells vary in size and shape, some of them being glia cells or ganglion cells, others being cylindrical in shape and representing the layer of rods and cones. The exact nature of the glioma is still a matter of doubt, but it is probably to be regarded as an endothelioma of the retina. (Fig. 239.)

The first thing to call attention to an eye affected with glioma is the presence of a gray or white reflex from behind the pupil. If the eye be carefully examined, it will be found that there are one or more white masses growing from the retina, containing bloodvessels. There is no pain; the eye is not congested. At a later stage the mass projects more forward until it fills the eye. Tension is usually raised during part of the time, and the eye becomes painful. In the third stage, the growth invades the optic nerve or finds its way out of the eye by other channels, where it forms masses which fill the orbit and produce great proptosis. The growth may find its way backward to the brain through the optic foramen; it may invade the frontal lobe of the brain by absorption of the roof of the orbit, or it may be reproduced in other distant organs of the body, chiefly in the liver. If left, the mass of glioma grows through the front of the eye, generally at the sclerocorneal margin, and forms a fungating, ulcerated, bleeding, painful mass. In its latest stages it produces death from exhaustion or by its attacking vital organs.

Fig. 239.



Glioma of the retina. (Leber.)

Glioma should be distinguished from purulent exudation into the vitreous—*pseudoglioma*. The absence of pain, tenderness, and inflammation in the early stage, the raising of the tension in the later stages, and the absence of retraction of the periphery and of the iris, help to distinguish it from pseudoglioma.

Treatment. The eye should be excised as soon as the disease is discovered. If this be done before the growth escapes from the eyeball, there is a good chance of cure. If the disease has advanced further, the orbit should be emptied, if possible, in order to save the child from suffering, produced by the fungating mass; but in such a case prognosis is very unfavorable.

Congenital Pigmentation of the Retina. A number of cases of pigmentation of the retina have been described by various authors. The pigmentation occupies a section of the retina only, and consists of collections of small round or angular masses of pigment grouped together somewhat like sarcinæ. They are unassociated with any choroidal change; they lie on the surface of the retina, and some-

times cover the retinal vessels. They have been considered as anomalous forms of retinitis pigmentosa, but they are not progressive, they do not accompany loss of function in the retina, and are probably of congenital origin.

Infantile Amaurosis. The history given by the parents in cases of infantile amaurosis is that the child was able to see well and noticed things, turned toward the light, grasped at objects held before it until the onset of complete blindness; this generally occurs under twelve months of age. The child may develop other signs of illness at the same time: general restlessness, feebleness of limbs and of back, or a condition of cervical opisthotonos. An ophthalmoscopic examination in some cases shows much dust exudation into the vitreous, with signs of syphilitic choroidoretinitis; in others optic neuritis due to tubercular meningitis may be present. But in many of the infants nothing can be seen by the ophthalmoscope at all, or a slight pallor only of the disk is seen after the blindness has continued for some time. It is in these cases that retraction of the head is most frequently met with. The cause is a posterior basic meningitis with distention of the ventricles of the brain by fluid. It usually happens that the blindness persists for some months and then recovery may take place, and even complete restoration of sight may follow. It is possible that the pupils may continue to respond to light during the whole attack, showing that the seat of disease is above the basal ganglia. An opinion is also sought by parents whose infants have never been able to see at all; in such cases the pupils may respond actively to light, and the optic nerve and retina are perfectly healthy. An examination of the head shows the skull to be very small in its upper part, the sutures to be prematurely united, and the fontanelles closed. Such children are microcephalic idiots, and no improvement is to be expected in their sight; the fault lies in imperfect development of the brain.

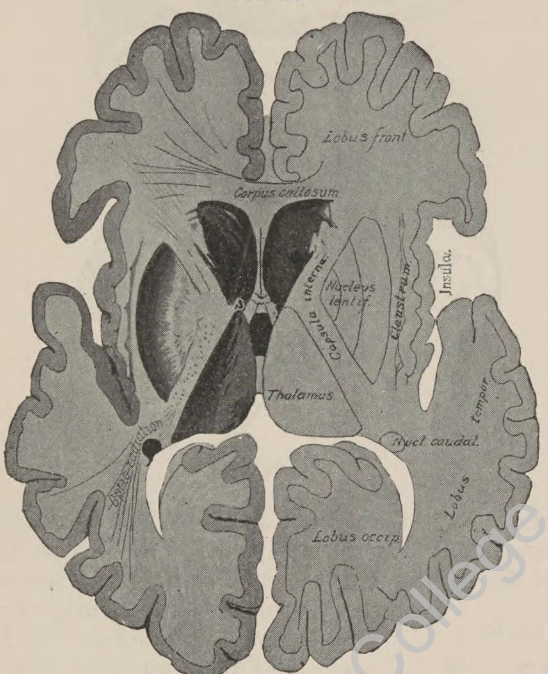
Treatment. The syphilitic choroidoretinal cases recover to a great extent under inunctions of mercury. The posterior basic meningitis cases recover if the health of the child is restored. The idiotic children do not gain any sight, and, although the condition of synostosis of the sutures has been met by craniectomy or removal of a portion of the roof of the skull, such measures probably do no real good.

THE OPTIC NERVE.

The optic nerve has its origin in the retina, passes through an opening in the choroid and sclerotic, the latter consisting of a fenestrated membrane known as the lamina cribrosa, traverses the orbit in a double curve in order to allow of free movement of the eye, passes through the optic foramen at the apex of the orbit and enters the skull. It is there joined by its fellow on the opposite side, to form the optic commissure or chiasma, where semidecussation of the nerves

takes place. The two halves of each nerve are continued backward from the chiasma in one cord, the optic tract, which winds around the crus cerebri and ends in the basal ganglia on each side. The basal ganglia are the external corpora geniculata, the anterior corpora quadrigemina, and the optic thalami. From these ganglia fibres pass in two main bodies to the oculomotor nuclei and to the cerebral cortex. The part of the cortex to which they are distributed is the mesial surface of the occipital lobe, the cuneus, and the neighborhood around the calcarine fissure. It is probable also that some of the optic nerve fibres pass on directly by the corona radiata to the occipital cortex, without entering the ganglia. (Figs. 240 and 241.)

FIG. 240.

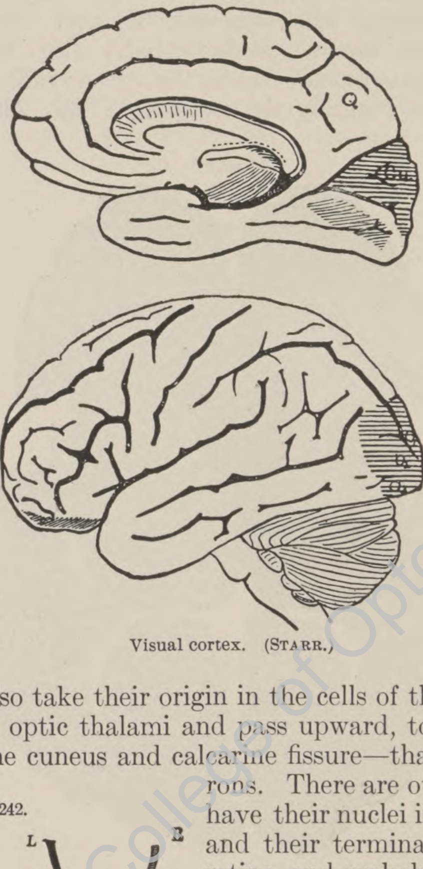


Optic radiations. (EDINGER.)

The Sheaths of the Optic Nerve. The coverings of the optic nerve are three in number, corresponding with the membranes of the brain. The dural sheath, continuous with the dura mater, forms a loose covering to the nerve, the pial sheath closely surrounds the nerve and sends septa to enter its substance. Between these two is the intervaginal space divided into two by the arachnoid. The fibres of the optic nerve at their entrance into the eye through the lamina cribrosa contain a medullary sheath; as they pass through this structure they lose their medullary sheath and are continued as translucent axis-cylinders only. Like the rest of the nervous system, the fibres

of the nerve are made up of neurons, the cells of which lie in the ganglion-cell layer of the retina, in close union with the visual epithelium, the layer of rods and cones. At their other ends the fibres end in brushes, chiefly in the external geniculate bodies and optic thalami. These are known as retino-thalamic neurons Visual fibres

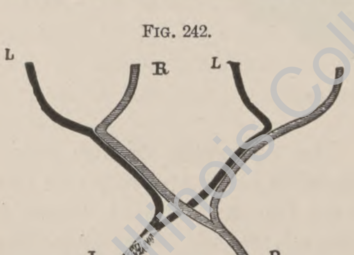
FIG. 241.



Visual cortex. (STARR.)

to the cortex also take their origin in the cells of the external geniculate bodies and optic thalami and pass upward, to be distributed to the region of the cuneus and calcarine fissure—thalamo-cortical neurons. There are other neurons which have their nuclei in the basal ganglia and their terminal branches in the retina, and probably some also which pass from the retina through the chiasma and optic tracts to the cortex direct. (Plate XVIII.)

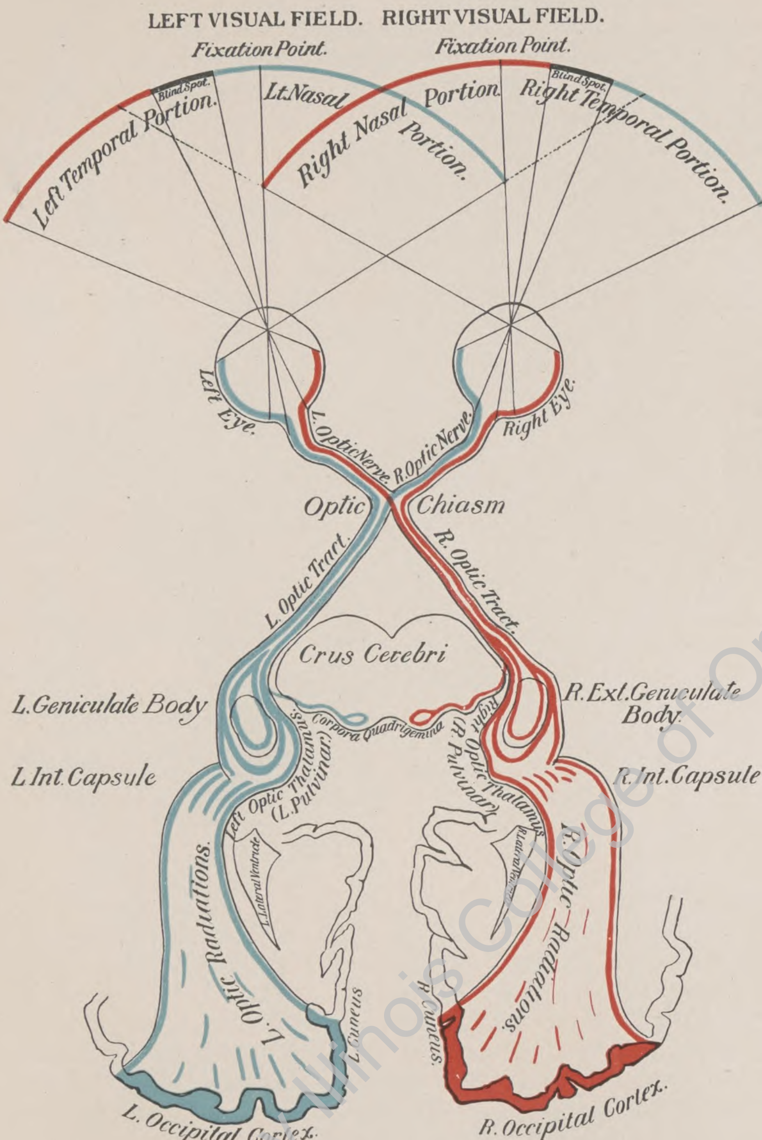
FIG. 242.



Decussation of optic nerve fibres. (WELLS.)

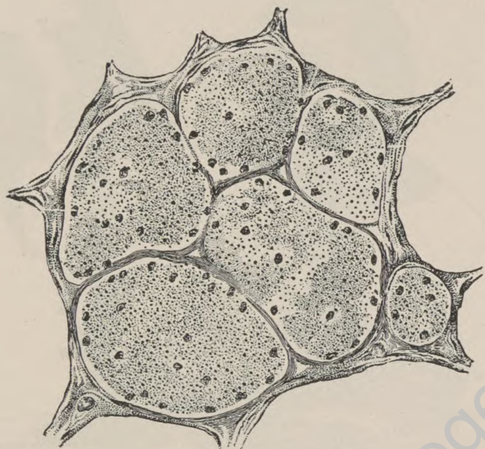
The chiasma lies in a groove at the base of the sphenoid bone in front of the infundibulum. In this commissure the optic nerves undergo a partial decussation. (Fig. 242.) The fibres from the right half of each retina meet in the chiasma and are continued on in the right optic tract; the fibres from the left half of each retina unite in the chiasma to form the left optic tract. The right optic tract

PLATE XVIII.


LEFT VISUAL FIELD. RIGHT VISUAL FIELD.
Fixation Point.
Fixation Point.
Blind Spot.
Blind Spot.
Left Temporal Portion.
Lt. Nasal Portion.
Right Nasal Portion.
Right Temporal Portion.
Left Eye.
Right Eye.
L. Optic Nerve.
R. Optic Nerve.
Optic Chiasm
L. Optic Tract.
R. Optic Tract.
Crus Cerebri
L. Geniculate Body
R. Ext. Geniculate Body.
Corpora Quadrigemina
L Int. Capsule
R. Int. Capsule
Left Optic Thalamus (L. Pulvinar)
Right Optic Thalamus (R. Pulvinar)
L. Lateral Ventricle
L. Optic Radiations.
R. Optic Radiations.
L. Cuneus.
R. Cuneus.
L. Occipital Cortex.
R. Occipital Cortex.

passes up to the occipital cortex of the right side, the left tract to that on the left side. From this it will be seen that the left half of the visual field in each eye is served by the right optic tract and right cortical visual centre; and the right half of the visual field in each eye is served by the left optic tract and left cortical visual centre. The division does not pass directly through the yellow spot; if one optic tract be destroyed, the edge of the blind area does not pass through the yellow spot, but leaves it intact in each eye. This is explained by the yellow spot being supplied by fibres going through each tract. As the nerve enters the retina, the most peripheral fibres supply the parts around the optic nerve, and the central fibres are distributed more to the periphery. The fibres of the nerve which supply the retina between the papilla and yellow spot, the papillo-macular bundle, are the most important, as they subserve the pur-

FIG. 243.



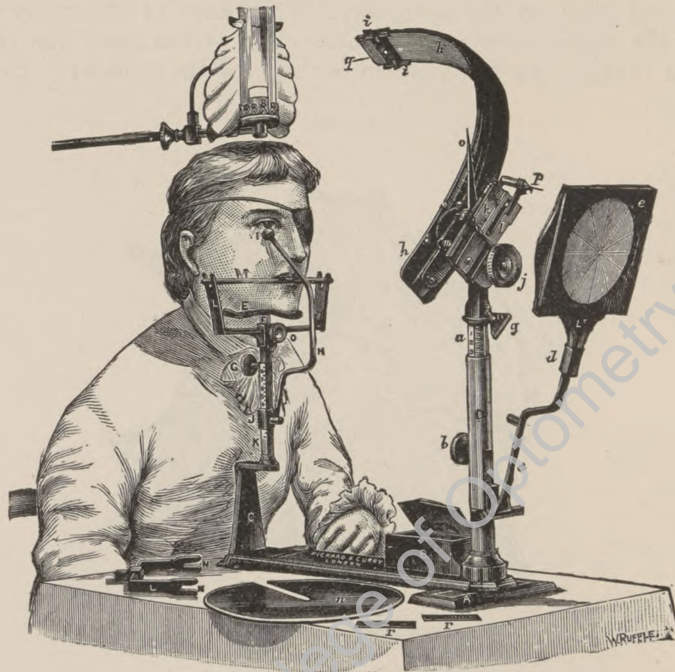
Section of optic nerve. (GRAEFE-SAEMISCH.)

poses of acute vision. Immediately behind the eye they occupy about one-third of the area of the nerve, in the form of a sector with its apex at the centre and base outward. Further back, these fibres lie in the axis of the nerve. From these anatomical arrangements we are able accurately to localize the seat of some lesions of sight. Thus, if one eye only be blind or defective, due to a nerve lesion, the seat of it must be anterior to the chiasma, while affections of vision of conjoint halves of the retina are due to disease of the tract or of the visual paths above it. Defects involving the fixation point, central scotoma, are due to diseases of the papillo-macular bundle.

In bitemporal hemianopsia the seat of the disease is in the chiasma. If one optic tract be affected, producing blindness of the same side of each retina, a condition known as homonymous hemianopsia, the pupils will not react to light thrown upon the blind halves of the

retina, but they will react to light thrown upon the seeing halves (Wernicke's hemiopic pupillary reaction). In this case, where the pupillary light reflex is interfered with, the lesion must be in the optic tract below the corpora quadrigemina, inasmuch as the path of the pupillary light reflex is from the optic tract to the corpora quadrigemina, thence to the third nerve nucleus, and outward along the third nerve to the pupil. If the pupils respond to light thrown on both halves of the retinæ, the lesion is higher up, either in the optic thalamus, internal capsule, or the cortex.

FIG. 244.



McHardy perimeter.

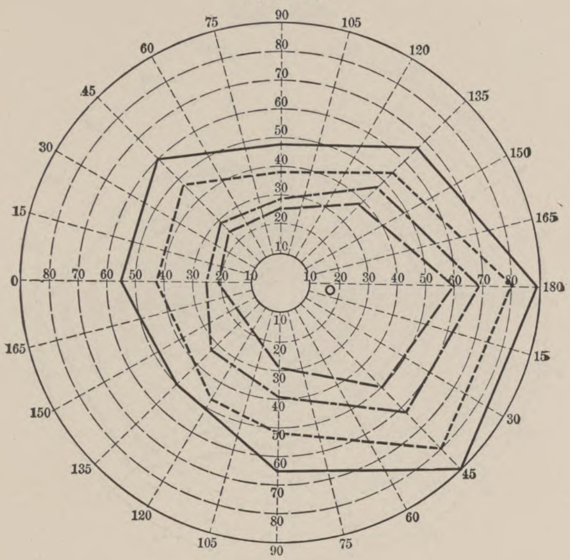
In diseases of the optic nerve the sight may be impaired in various ways, central or peripheral vision may be interfered with, the vision in each eye may be lost, or the perception of colors may be destroyed.

Peripheral vision implies the perception of objects all around the point directly looked at. Thus, if we cross a street, although we may be looking directly in front of us, we are conscious of the movement or approach of vehicles on each side of us. We are also able to appreciate generally the quality of the surface on which we are walking, and to avoid obstacles in our path without directly looking at them. If this power were absent, as it is in some diseases, we should be in the position of a person looking down a long tube; it would be difficult for us to find our way about; all power of orientation would be lost.

The whole area from which the eye is capable of receiving impressions is called the field of vision, and it is capable of being measured

FIG. 245.

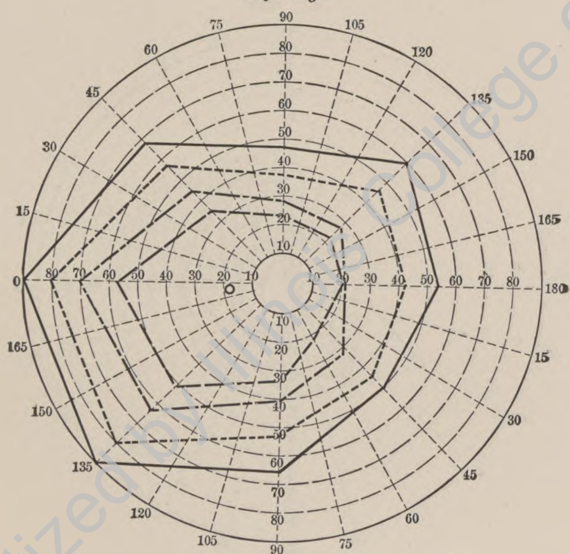
Right Eye



Normal field.

FIG. 246.

Left Eye

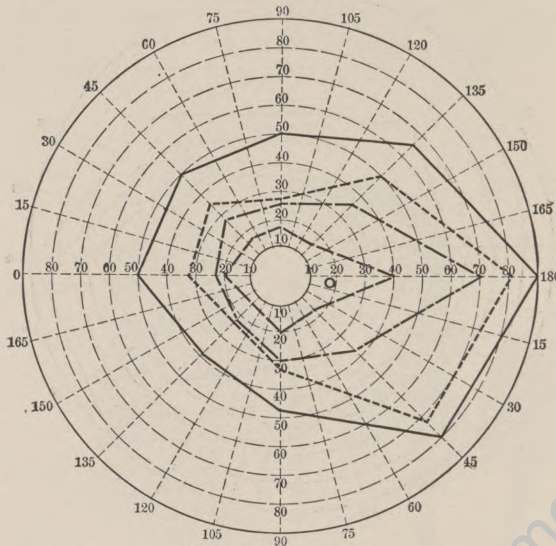


Normal field.

in several ways: by the hand, by light moved before the eye, or more accurately, by an instrument called the perimeter. (Fig. 244.) In measuring the field by the hand, the patient is placed with his back

FIG. 247.

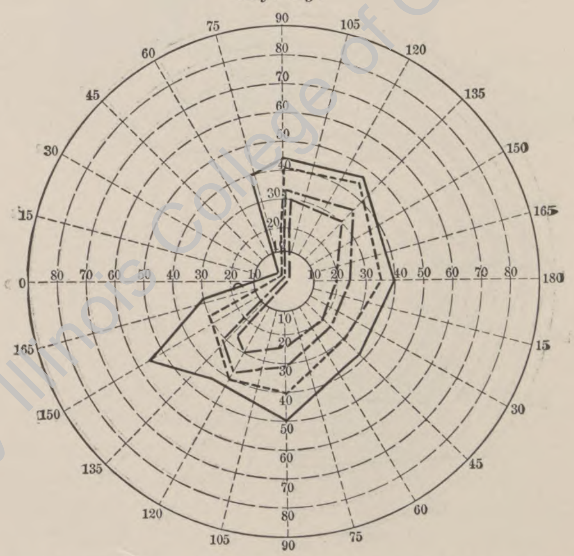
Right Eye



Eccentric contraction of field as seen in gray atrophy of the optic nerve.

FIG. 248.

Left Eye

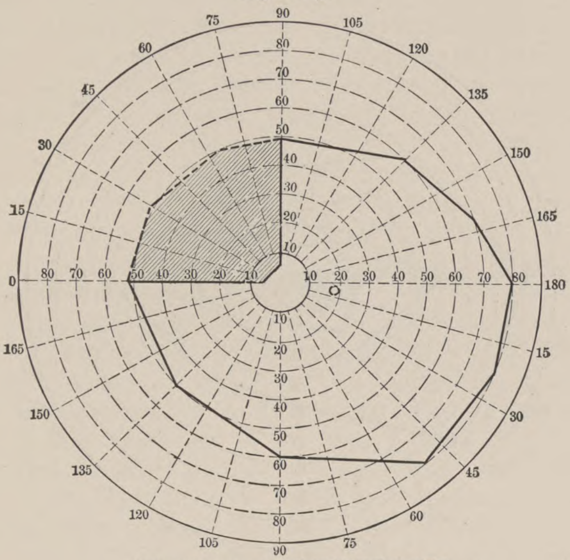


Eccentric contraction of field as seen in gray atrophy of the optic nerve.

to the light, the hand held in various positions before him, and he is asked to point out its direction, at the same time keeping his eye fixed on the observer's face, directly in front of him. A small piece

FIG. 249.

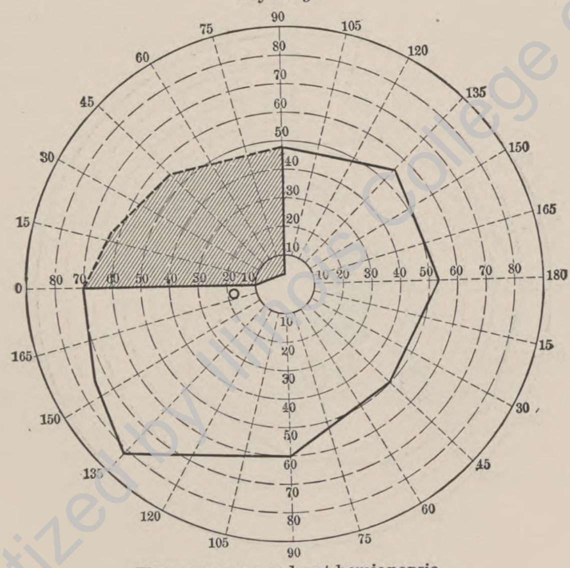
Right Eye



Homonymous quadrant hemianopsia.

FIG. 250.

Left Eye

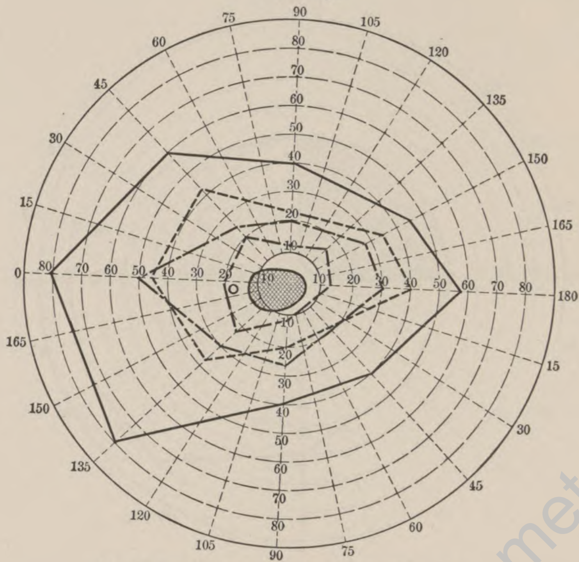


Homonymous quadrant hemianopsia.

of white paper may be used with the same object. If the sight is impaired by disease of the front of the eye, we can get some knowledge of the condition of the visual field by holding a candle in

FIG. 251.

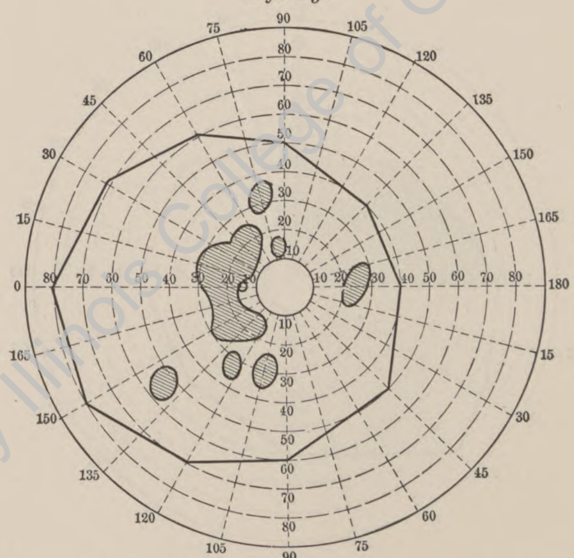
Left Eye



Central scotoma as seen in toxic amblyopia.

FIG. 252.

Left Eye



Scotomata as seen in disseminated choroiditis.

various positions before the eye, or by throwing upon the eye the light reflected from an ophthalmoscope mirror—the projection test.

The perimeter consists of a quarter or half-circle of metal, revolving around a fixed point, at which is placed a small white spot, the object to be looked at by the eye under examination, the fixation point. The eye is placed at the centre of the circle, and another white spot is made to travel along the circle from the fixation point until it can no longer be seen; the point of its disappearance is the limit of the visual field in that direction. In practice it is customary to start with the travelling spot at the extreme periphery, and to mark as the outer limit of the visual field the place at which it first becomes visible as a distinct spot of white This limit is a constant one in healthy eyes. The visual field extends about 95° to the temporal side, about 60° upward, 50° inward, and 80° downward. (Figs. 245 and 246.) The limit upward and inward varies with the prominence of the brow and nose, but it is, apart from this, less than in the temporal direction. The size of the travelling spot used varies according to the degree of affection of sight. It is well to use as small a spot as can be seen with ease for this purpose; 5 mm., 2½ mm., or even smaller spots may be used. But where the acuteness of sight is much reduced, it is necessary to use spots 10 mm., 15 mm., or 20 mm., in diameter. In doubtful cases it is also desirable to take the field with diminished illumination. The color fields may be taken in the same way as the field for white by using a small colored object instead of a white one. The size of the color field varies with the size of the object and the brightness of the illumination. With very bright light and a sufficiently large mass, color can be recognized at the extreme periphery of the visual field, but with small-sized objects the periphery of the retina is incapable of appreciating their color. The field for blue is the next in size to white, then follows red, and, lastly, green. It is important to take the color fields in some cases of optic nerve disease, as the test is a more delicate one than that for a white spot, and often indicates very early stages of optic nerve atrophy. (Figs. 247 and 248.) Defects in the field of vision may take the form of a concentric contraction, or they may be limited to one portion of the field, such as a sectional area triangular in shape, with its apex at the centre, its base at the periphery (Figs. 249 and 250); or there may be gaps in the field or blind spots of various shapes. These are known as scotomata, and may be either at the point of fixation—central scotoma (Fig. 251); or outside it—paracentral scotoma. They may form a blind ring around the fixation point—ring or annular scotoma; or they may be situated in other parts of the field, where, as a rule, they are of little practical significance. (Fig. 252.) It should be noted that the entrance of the optic nerve into the eye, inasmuch as it contains no retinal elements, is a blind spot. It is placed about 15° outside the fixation point. (Figs. 245 and 246.)

Scotomata may be either positive or negative: positive when they form a dark spot in the field of vision, which the patient is conscious

of; and negative when they form merely a gap in the field which is blind, but which is not objectively present as a dark area to the patient. Again, scotomata are either absolute or relative: absolute when all perception is lost, relative when perception is merely dulled. Thus, a scotoma is said to be absolute when all perception of light and form is lost in it; relative, when there is loss of perception of color only.

The Light Sense. (See page 35.)

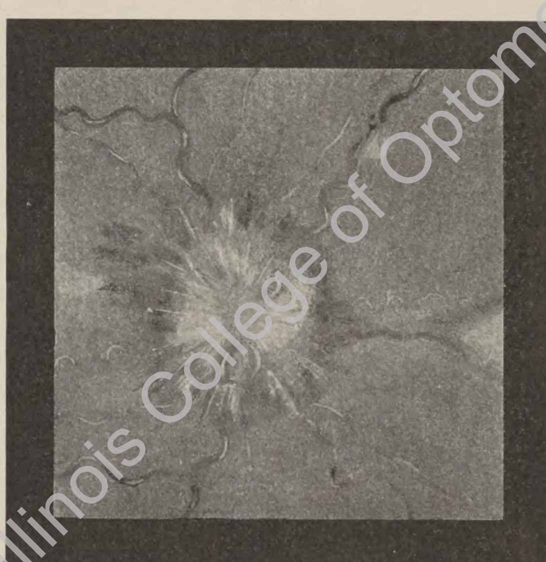
Congenital Peculiarities.

Coloboma of the Sheath of the Nerve. This condition, due to imperfect closure of the fœtal cleft in the nerve, is sometimes associated with coloboma of the choroid and sometimes occurs independently. It appears as a very large and deep excavation of the lower part of the nerve, the whole disk being sometimes very much enlarged.

Opaque nerve fibres are seen on the disk alone sometimes, but they generally spread out over the retina. (See Retina.)

Pigmentation of the Nerve. The optic nerve is sometimes very highly colored, of a dull red-gray hue; this condition, which may be mistaken for optic neuritis, is congenital.

FIG. 253.



Acute optic neuritis. Note disk much swollen: estimated at + 7 D. Edge indistinct; vessels obscured at edge; large number of hemorrhages around the disk, patches of œdema in the retina, veins very tortuous.

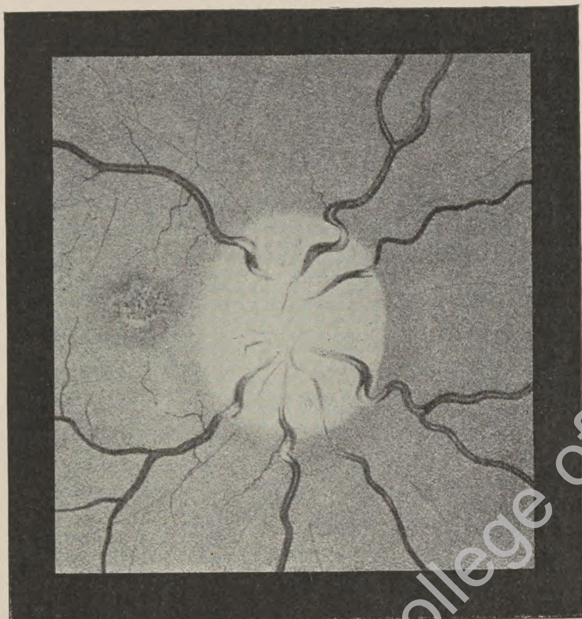
Inflammation of the Optic Nerve: Optic Neuritis. Inflammation of the optic nerve may take place at any point in the course of the

nerve. If the intra-ocular portion is affected, the disease is spoken of as papillitis; but if the trunk of the nerve only is affected, the inflammation does not show itself ophthalmoscopically in the head of the nerve, and it is known as a retrobulbar or retro-ocular neuritis. (Fig. 253.)

Papillitis. Inflammation of the Head of the Optic Nerve. This shows itself in two main forms, but there is no sharp line of division between them, and many cases have characteristics which will bring them under each heading.

1. The swelling is sharply limited to the disk; in the earliest stages the edges of the disk appear blurred; the natural striation of the retina

FIG. 254.



Swollen disk in a case of chronic meningitis. (LIEBREICH.)

at the edge of the disk is more marked; at the same time the disk itself becomes redder in color, the veins are full and show tortuosity, with a tendency to disappear into the œdematous retina at the edge of the disk. (Fig. 254.) The vessels as they emerge from the central pit appear to come markedly forward, and a movement of parallax is obtained against the background of the nerve. By this test, if the vessels be kept in view while the head of the observer is made to move a little from side to side, they will appear to move against the edge of the disk behind them, showing that they are not in contact with it, but lie at some distance in front of it. As the disease advances the papilla becomes more and more prominent and stands out into the vitreous. The nerve may be increased in redness, or

it may become pale from pressure on it, and may contain masses of exudation. The arteries are small, the veins very full and tortuous; where the vessels pass over the edge of the disk they may disappear into the œdematous retina or behind the prominent and overhanging head of the nerve. The amount of swelling should be estimated by the direct method with the ophthalmoscope. At the same time numbers of retinal hemorrhages may appear around the disk. The retina between the disk and yellow spot is frequently œdematous, and is thrown into folds radiating from the yellow spot and containing degenerated products of inflammation similar to those that have been described in albuminuric retinitis. As the inflammation subsides, the vessels gradually regain their normal appearance. The swelling and œdema disappear; the disk is left permanently pale, but signs of past inflammation show themselves in the edge of the disk and along the vessels. This variety of optic neuritis is known as *choked disk.*

2. In the other form, *descending neuritis*, the inflammation is not confined strictly to the papilla; it extends for some distance into the retina. Thus we find a small degree of swelling, but more œdema of the retina and more hemorrhages with white areas of exudation into the retina. It is difficult sometimes to decide from the ophthalmoscopic examination alone whether we have to deal with an albuminuric retinitis or with an optic neuritis due to intracranial disease. But, as a rule, there is less swelling of the optic nerve in albuminuric retinitis than in optic neuritis. The urine should be examined, as albumin is not generally present in optic neuritis.

Causes. Optic neuritis may be caused by a local disease in the brain or by some general poison. The local disease is generally of an irritating kind: meningitis or a tumor. The meningitis is either acute or chronic; it may be due to middle-ear disease or septic thrombosis in the cavernous sinus, to abscess of the brain, to tubercle, generally in the form of miliary tuberculosis of the meninges in young children; it may be due to posterior basic meningitis caused by pneumococcus infection, or to hydrocephalus. It is caused in rare cases by spinal cord disease, such as acute myelitis or cerebro-spinal meningitis. The tumors include intracranial growths of every kind, gliomata, sarcomata, tuberculous masses. Gummata are the most common causes of optic neuritis. In about 80 per cent. of cerebral tumors optic neuritis is present, although its occurrence may be late in the course of the disease. The seat of the growth bears no relation to the extent or duration of the disease; thus, a growth in the spinal cord may give rise to a well-marked papillitis, whereas extensive gliomatous change in the frontal lobes may exist for a long time without producing optic neuritis. Non-irritating diseases, such as hemorrhages, cysts like cysticercus or aneurism, are not generally followed by optic neuritis. The presence of a gumma in the brain not in connection with the optic nerves and acting after the manner of a tumor may produce neuritis. A gumma may also be formed in the

optic tracts or chiasma directly, or it may in some cases form in the head of the optic nerve itself—*syphilitic papillitis*.

The neuritis is generally double, but inflammation about the apex of the orbit, the optic foramen, the sphenoidal fissure, erysipelas of the head extending to the orbit, or distention of some of the fossæ of the nose pressing on the nerve, may give rise to an optic neuritis confined to one side only.

The general or systemic causes of optic neuritis may be pneumonia; exanthematic fevers, such as influenza, typhoid, measles, scarlet fever, etc.; severe anæmia, lead-poisoning, suppression of menstruation, post-partum conditions, or sudden loss of blood.

Course and Symptoms. The optic neuritis may exist for a long time without discovery, the failure of sight being often so slight as to be unnoticed by the patient. If the attack is rapid and not severe, the optic nerve may recover and only show by the ophthalmoscope that it has passed through a condition of neuritis. On the other hand, the failure of sight is sometimes very sudden and complete. In one case the failure was so sudden that the patient complained that someone had turned down the gas. Failure may go on to complete blindness, and yet recovery may take place; in other cases vision fluctuates very much. Occasionally cases are met with presenting all the signs of cerebral tumor: headache, vomiting, optic neuritis; the optic neuritis may subside, leaving the disk more or less atrophic and the vision impaired; the other symptoms may cease and the patient may be restored to a condition of perfect health. Others again, especially children, pass through a very severe attack of double optic neuritis without the general health appearing to suffer in any way at the time, and recover with permanently pale disks, and never have another attack. It is probable that the cause of the neuritis in many such cases is tubercular meningitis, which has been recovered from, or a mass of tubercle lying in the brain, shut off from the tissues around it. A temporary disturbance in the mass of tubercle sets up an inflammation around it, produces neuritis, and then rapidly subsides, giving no further trouble. In other cases the quiescence is not permanent, but recurrences of cerebral irritation follow at long intervals, the patient being in good health between them. Each attack corresponds to a period of advance in some slowly growing cerebral neoplasm. One such case was that of a girl of about eighteen, who is still under observation, who has been known to have had optic atrophy following neuritis for ten years. She has periods of good health, and then attacks of very intense pain in the head, vomiting, delirium, etc., recurring at intervals of several months. In one of these attacks she had violent epileptic fits, which produced petechial hemorrhages of the conjunctiva and face. The disks are quite white and she is practically blind; although she has central vision of $\frac{6}{18}$, it is at such a pin-point area in the centre of the blind visual field that it is with the utmost difficulty that she can find the object she wishes to look at.

The attacks vary very much in their duration; some of them are very rapid and pass away entirely in a few weeks, ending in recovery with or without destruction of sight; in others the condition is very chronic, the appearance of neuritis in the disk being present for months. As a rule, attacks are single, but cases have been described by Anderson, Gowers and others in which second attacks have been observed, and Gunn has seen optic neuritis occur in a well-developed form in a disk which had previously been noted to be atrophic. Optic neuritis occurring during or after pregnancy is probably due to some toxic condition arising from the uterus. It subsides after a time without much impairment of vision.

Anæmia may give rise to the most severe optic neuritis and very great swelling of the disk. This may be a simple swelling consisting mainly of œdema, or it may be accompanied by large numbers of retinal hemorrhages and exudations into the retina. Optic neuritis due to anæmia is often very sudden in its onset. It presents a contrast to that occurring in cerebral tumor. In addition to making an examination of the general state of health of the patient, with analysis of the urine, it is advisable here to make an examination of the blood, counting the number of corpuscles.

Suppression of menstruation from exposure to cold is said to cause rapid failure of sight after optic neuritis. It is possible that many such causes may come under the heading of anæmic or chlorotic neuritis.

Optic neuritis in lead-poisoning varies in degree from the slightest haze of the disk to a severe acute swelling of the disk with hemorrhages. It sometimes extends widely into the retina, producing an ophthalmoscopic appearance not much distinct from albuminuric retinitis of granular kidney. In this case it is probable that the cause lies in the granular condition of the kidneys, caused by Bright's disease, which is very likely to occur in those suffering from lead intoxication. The urine should be examined, and other signs of lead-poisoning sought for, such as the presence of a blue line on the gums.

Spurious Optic Neuritis. Mention must be made here of a condition of the disk, which is sometimes met with, resembling optic neuritis in the acute stage. The disk is red, congested, with blurred edges, and a measurable amount of swelling, or it may appear blurred and rather pale, as in a subsiding neuritis. The vision of the eye is normal, the visual fields are normal, the color vision and the light sense are normal, and there is no history of any previous defect of sight. Many of these cases have been watched for years and no change has been noticed in the ophthalmoscopic appearances. A smaller degree of the same condition is not uncommonly met with in the red and streaked disk of hypermetropia. In both of these conditions the appearance of neuritis is probably due to a congenital peculiarity of the optic papilla.

Cause. The causes of optic neuritis in their relation to cerebral tumor have been discussed at great length by many writers. Von

Graefe explained it as being a swelling produced by a blocking of the cavernous sinus and of the tributary ophthalmic vein, hence the name "choked disk." Schmidt-Rimpler and Manz showed that the fluid displaced by a cerebral tumor found its way into the lymph spaces of the optic nerve, the intersheath spaces became distended with fluid, especially at the ocular end, where there is an ampulla-like enlargement of the intersheath space. The pressure of this fluid passed into the optic nerve and compressed the retinal vessels, by which the thin-walled veins were affected more than the arteries. Hence, a hindrance to the exit of fluid was produced, which gave rise to the choked disk appearance. According to Leber, the presence of this fluid sets up a toxic condition and gives rise to an inflammation of the nerve. Other writers have held that the neuritis is a true descending one; they have shown that there is an increased cellular exudation in the tissues surrounding the cerebral tumor, which extends the whole way from the tumor to the nerve and along the latter to the eye. Whether this be the fact or not, there can be no doubt that a great deal of the swelling in choked disk is caused by the pressure of fluid in the cranial cavity, and secondarily in the subarachnoid space of the nerve, inasmuch as relief of pressure causes reduction of the swelling in the disk. The results of Horsley's work have shown that trephining the skull in cases of cerebral tumor causes diminution in the optic neuritis, even when it has been found impossible to remove the tumor.

Prognosis and Treatment. The prognosis depends very much on the nature and cause of the neuritis. In a case of cerebral tumor which is incapable of removal, and which is steadily growing, the prognosis is serious; the treatment is that of the cerebral tumor. But even if the tumor be necessarily progressive and incapable of removal, life may still last many years, and, if the sight can be saved, it should be done. It may be said that the operation of incising the distended sheath of the nerve behind the eye offers no prospect of relief, but the results of Horsley's work give great hope of lessening and even of stopping the optic neuritis by trephining the skull and relieving intracranial pressure. In suitable cases this plan should be carried out; it is scarcely necessary to use it where the optic neuritis is not very severe and does not interfere much with the vision. Much reduction in the amount of swelling can also be sometimes obtained by the use of iodide of potassium. In chlorosis the prognosis is good, but it depends on an early recognition of the cause. Treatment should be in the main by iron, aided sometimes by arsenic and by regulating the other factors of health, especially by relieving constipation. In cases associated with disorders of menstruation hot baths should be given and leeches applied to the temple. In the acute specific fevers the prognosis is good and treatment calls for no special remark. In lead-poisoning the prognosis is not very good; the cases go on frequently to atrophy, and the possibility of renal complications arising should be borne in mind. The treatment is

that of lead-poisoning generally. The cases occurring in pregnancy recover without going on to complete blindness; they require no special ocular treatment. The syphilitic cases should be treated according to the seat of the syphilitic lesion. If it be a local affection of the head of the nerve, syphilitic optic neuritis, inunction of mercury should be used; if this be carried out efficiently in the early stage, the prospect of recovery is good. In the other class in which the optic neuritis is secondary to gummatous disease of the brain, the prognosis is less good and the treatment should be in the main by iodides.

Retrobulbar Neuritis. Retrobulbar neuritis, the other main form of inflammation of the optic nerve, as distinguished from papillitis, shows itself by changes in the nerve behind the eye, and only appears in the papilla at a later stage. It produces a diminution of central vision, the general area of the visual field being unchanged. It has been investigated by many observers, of whom the earliest were Samelsohn and Nettleship. The results of their investigations showed that it was the papillo-macular bundle of fibres employed in central vision which was affected. The papillo-macular fibres supply the yellow spot region, and in the optic nerve occupy the temporal side of the disk. In the anterior part of the nerve behind the eye they form a wedge-shaped segment, whose apex is toward the centre, and base toward the temporal border of the nerve. Further back in the nerve these fibres occupy a more central position and do not reach the edge of the nerve; in the skull they form an oval mass below and to the temporal side of the centre. In inflammation of this bundle of fibres there are proliferation of the cells of the neuroglia, engorgement of vessels, and interstitial neuritis, and at a late stage degeneration of the axis-cylinders from pressure. The effect on vision of this inflammation of the papillo-macular fibres is the formation of a blind spot in the visual field, extending from the nasal side of the point of fixation longitudinally outward as far as the normal blind spot. The density of the scotoma varies from an inability to distinguish color or a lessened perception of color at the centre—relative color scotoma—to a loss of perception of form—absolute scotoma. The causes may be classified according to their seat and nature into: Local causes—orbital cellulitis, erysipelas, local periostitis, syphilitic or not; tubercle or septic inflammation from the neighboring sinuses; of these, the sphenoidal sinus is the most likely one to give trouble, as it is separated from the optic nerve by only a thin layer of bone. General causes: gout, diabetes, the various poisons, general septicæmia, hereditary retrobulbar neuritis, and disease occurring in the optic nerve as part of the nervous system generally. The disease shows itself in an acute and a chronic form.

Acute Retrobulbar Neuritis. This is manifested by a rapid failure of sight on one eye, generally preceded by neuralgic pain in the temple, pain in pressing the eyeball back into the orbit, and in movement of the eye from side to side. As a consequence of the pain elicited

by movement of the eyes, they are frequently kept closed. At first there is scarcely any ophthalmoscopic change, but later on the disk becomes paler and the vessels may become contracted, the pupils are dilated and somewhat inactive to light; or if the reaction is good, it will be found that the contraction on exposure to light is not maintained, the pupil quickly rebounds.

Failure of sight goes on increasing for four or five days, reaches its height, and, after a week or so, begins to recover slowly, the period of recovery occupying a month or six weeks. The defect is often described as a mist or dark spot which covers the objects looked at, and gives the appearance of a gray-brown spot on a white surface. The vision is worse in bright light and improves after rest, as in the early morning after a night's rest. This effect of bright light is probably caused by over-stimulation of the weakened nerve elements or by their imperfect insulation. It was noted by Berry as a point of difference between papillitis and retrobulbar neuritis that the light difference, as tested by Bjerrum's types, was scarcely present at all in papillitis, but was strongly marked in retrobulbar neuritis. The visual fields are not contracted in the ordinary cases, but there is a central defect of vision extending from the yellow spot and including the optic nerve entrance. Sometimes, however, central vision does not fail, but a peripheral contraction of the visual field may be present, as in the case of periostitis in the optic canal, when the defect in the visual field corresponds to the seat of pressure on the nerve. Hock believes that it is possible to indicate the seat of the disease by the direction of the movement of the eye, which produces pain, this being due to stretching of the sheath of the nerve. Thus, pain on looking up would indicate the lower part of the sheath of the nerve as the seat of the inflammation, and the upper part of the visual vessel as contracted. This is certainly not always true. After recovery has set in, the process may continue until the sight is quite restored to the normal, or there may be permanent loss of vision. In those cases where the focus of the disease is close behind the eye, the papilla is involved, and it is not easy to distinguish the case from one of ordinary papillitis. In ordinary papillitis the failure of sight comes on later in the disease, even after the attack has begun to subside, while in retrobulbar neuritis, failure of sight occurs at the very beginning.

Treatment. In addition to the treatment of whatever may be the obvious underlying cause of neuritis, such as syphilis, tubercle, gout, etc., much may be done in the way of local measures. Dark glasses or goggles should be worn in bright light; leeches or the artificial leech, or blisters should be applied to the temple and all use of the eyes forbidden. Iodide of potassium should be given and any local disease in the nose should be dealt with.

Chronic Retrobulbar Neuritis. Under this heading we find the various forms of toxic amblyopia, the most common of which is that produced by tobacco or tobacco combined with alcohol. Other

causes are bisulphide of carbon, used in the curing of vulcanite, iodoform, occasionally seen after its use in surgery; nitrobenzol, dynamite, methyl alcohol, lead, male fern, pomegranate root, Jamaica ginger, and other substances.

The question of the existence of pure alcoholic amblyopia appears now to be established in the affirmative, but for a long time it was a much disputed point, for no case of amblyopia was met with in which there was an entire absence of the use of tobacco. Alcohol does undoubtedly frequently influence the course and nature of the tobacco amblyopia. The worst cases met with and the ones which result in imperfect recovery are those in which considerable amounts of alcohol have been taken in addition to the use of tobacco.

Diabetic Amblyopia. The question of amblyopia has also been discussed from the point of view of its association with diabetes as a cause. Undoubtedly diabetics are very susceptible to the influence of tobacco, which is apt to produce in them a toxic amblyopia. The rapidity with which this may be brought about is shown by the case of a patient, aged fifty-four years, who had been known to have diabetes for at least two years. About four months before he was first seen he had retired from active work, and, time being heavy on his hands, he had taken to smoking for the first time in his life. He smoked very moderately—not more than one pipe a day—and his sight began to fail about three months after beginning to smoke and about one month before he was seen. His sight at that time was R. $\frac{6}{60}$, L. $\frac{6}{60}$. He had a central color scotoma, no contraction of his visual fields; his optic disks were rather pale. He at once discontinued tobacco, but his sight continued to fail, and two months later was reduced to $\frac{2}{60}$ in each eye. His optic nerves were then very pale. Although most of the diabetics who have come under observation with central scotoma have been users of tobacco, a few cases have been met with, some of them among women, in which there appeared to be no cause for amblyopia apart from the diabetes itself.

Pathology. The changes that have been found post-mortem have been confined to the papillo-macular fibres of the optic nerve. They consist in thickening of the coats of the vessels in the optic nerves and an interstitial neuritis or increase of the connective-tissue elements in the optic nerve with atrophy of the nerve fibres. Lately opinion has been leaning toward the view that the change begins by a degeneration in the macular region of the retina. The experiments of Langley on the influence of nicotine on the activity of the ganglionic cells has further stimulated inquiry in this direction. Nuel has held from examination of a section of the yellow spot that the disease starts in the macula lutea. Whether this change is due to the action of nicotine, or, possibly, some of the other constituents of tobacco smoke, notably pyridine, on the ganglionic cells or their synapses, or whether the changes found in them are due to an interference with their blood supply, must, for the present, remain undetermined.

Symptoms. There are sometimes present the symptoms of general tobacco-poisoning, rapidity of the heart-beat, sleeplessness, loss of appetite, tremors, etc.; but these may be conspicuously absent. The sight is dim, there is a mist or a fog over the objects directly looked at, and an inability to distinguish colors, as, for instance, to tell readily a sovereign from a shilling. The sight is worse in a bright than in a subdued light; the acuteness of vision may be considerably reduced, from $\frac{6}{6}$ to $\frac{4}{60}$, or even less. Ophthalmoscopic examination shows slight haziness of the disk generally, with pallor of the temporal half. In other respects the fundi are normal, and even these changes are by no means constant or easily recognized. The visual fields in true tobacco amblyopia are of normal extent. But at the fixation point there is a scotoma or blind area for colors, extending from the fixation point to the blind spot. The size of the scotoma and its density vary very greatly; its average size is an oval, having a horizontal diameter of about 20°, with a vertical diameter of 12°. Sometimes it is larger and extends outward almost to the limit of the color field, when it is sometimes difficult to discover the nature of the scotoma, unless we use test spots of considerable size. Perception of green is lost first, then of red; perception of blue and yellow are less frequently lost. The point of greatest saturation of the color defect lies outside the fixation point. It is nearly always possible to make out at this spot that the scotoma is absolute—that is, that perception of everything is lost at it—but the examination requires care with a very small test object. It is sometimes difficult to obtain good evidence of the scotoma, because of the inability of the patient to fix the object steadily. This is most commonly found when the condition of tobacco shakiness is far advanced, and especially when it is complicated with chronic alcoholic poisoning. The shiftiness of the eye under observation and the wandering attention of the patient are well marked and almost characteristic. If this defect be met with, the best method of making the examination is to cut out a piece of paper, red on one side and green on the other, about 5 mm. square, and stick it into the nib of a pen. Armed with this, stand directly in front of the patient, and tell him to look steadily at the point of your nose, one eye being covered. It is possible then to change rapidly the position of the color spot, and, at the same time, to keep a close watch on the fixation of the eye. In this way the examination is made accurately and rapidly. If it is desired to keep a record of the size of the scotoma, it should be charted on the perimeter. In addition to finding the scotoma, it is necessary to see that the field of vision is not contracted, that the pallor of the disk is limited to the temporal side, that there are no other signs of nerve disorder, like tabes or insular sclerosis, before coming to a conclusion as to the nature of the disease.

Prognosis and Treatment. The prognosis is good, provided that the patient will abstain from all use of tobacco; improvement may follow diminution of the amount used, but it is well to insist that all

tobacco should be given up, as a very small amount is often sufficient to keep up the irritation. In addition, it is well to give small daily doses of strychnine. Improvement generally sets in after about two weeks and goes on to complete recovery. As to whether the patient may resume smoking after recovery or not, he may do so to a very moderate degree after an interval of months; but, seeing the hold that tobacco has on many confirmed smokers, it is well, when the habit has been broken, for it not to be resumed, for relapses, although uncommon, may occur. In some of the worse cases of tobacco-alcohol blindness the loss of vision amounts to all but a perception of light. In such cases strychnine should be given in full doses. It is useful to combine it with nitroglycerin or to give inhalations of amyl nitrite; at the same time the general health must be considered, as such patients are often broken down, and sometimes on the brink of delirium tremens. Sleep and a sufficient amount of nourishing food must be secured.

Bisulphide of Carbon. The symptoms of bisulphide of carbon poisoning resemble much those of the most acute of the tobacco-alcohol cases. They are giddiness, pallor of face, unsteadiness of gait, and tremors, with failing sight, a mist before the eyes, dilated pupil, and ophthalmoscopically disks which are pale or hazy all over. The effect of carbon bisulphide is more severe than that of tobacco. In a collection of cases made by the Ophthalmological Society of the United Kingdom, 33 per cent. recovered vision, 25 per cent. improved, and 20 per cent. did not improve at all.

This disease is very rare now, owing to improved methods of ventilation in factories, by which the vapor is not allowed to circulate among the workers, but is drawn out of the room by proper extractors, so that cases rarely come under care. Treatment is that of tobacco amblyopia.

Chronic lead-poisoning is characterized, in addition to the general signs of plumbism, by loss of sight, often of the central scotoma type, slight chronic neuritis of the optic nerve, passing on to atrophy, with some contraction of the field of vision. In addition there may be the signs of albuminuric retinitis secondary to granular kidneys, caused by lead-poisoning.

Hereditary Optic Atrophy. This disease, which is characterized in its onset by loss of central vision, the periphery of the visual field being retained, probably belongs to the retrobulbar neuritis group, although the ophthalmoscopic appearances, as a rule, are those of simple atrophy. It begins in early adult life, generally from eighteen to twenty-five years of age, attacks the male members of a family mostly, is transmitted through the females, and generally appears in successive generations. Another feature of the history of these families is the large number of early infantile deaths which it reveals. The disease is generally slow in its progress and is unaffected by any treatment. It does not go on to complete blindness, a certain amount of peripheral vision being retained.

Amblyopia is said to be produced by a very large number of agents. But such causes are very uncommon; the only one which calls for special mention is

Quinine Amblyopia. This may be caused in susceptible individuals by comparatively small doses of the salts of quinine; but the doses known to have caused blindness are from 15 grs. to ʒj, taken in the day. The loss of sight comes on rapidly and varies in degree, but it may be total. The pupils are dilated and irresponsive to light; ophthalmoscopically the disks are seen to be pale, the retinal vessels very small, like the appearances in atrophy of the optic nerve. Œdema of the retina is sometimes present, and a cherry-red spot at the macula, like the appearances in embolism of the central retinal artery, is met with. It can sometimes be recognized that the visual fields are strongly contracted. Recovery takes place first at the centre of the field for form and then for color, but a certain amount of contraction of the fields of vision often remains permanently.

Prognosis. In most cases recovery takes place to a certain extent, but the process goes on for months before it is complete. Relapses may occur if the drug be again administered. The experiments of Brunner, Barabaschew, and de Schweinitz have shown that the lesion is a peripheral one, depending on defective nutrition of the nerve and retina, from extreme contraction of the retinal vessels. There is no neuritis, but thickening and obliteration of the lumen of the arteries supplying the optic nerve, chiasma, and optic tracts are seen. According to the experiments of Ward-Holden, in dogs fed on quinine the change first shows itself in the ganglionic cells of the retina and nerve fibres; by the forty-seventh day the ganglionic cell layer and nerve-fibre layer had almost disappeared. He holds that with a lessened blood supply the less resistant elements of the retina—the ganglionic cells—break down, and that there is an ascending degeneration of the nerve fibres secondary to this change in the nerve cells.

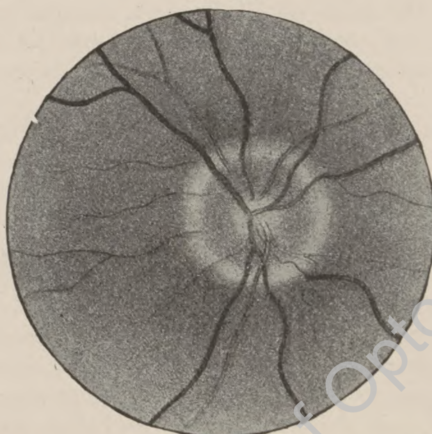
Treatment. Administration of quinine and its salts must be stopped. Nitrite of amyl or nitroglycerin internally are the best forms of treatment. Strychnine, digitalis, and iodide of potassium may also be used.

Retrobulbar Neuritis Due to Disease Affecting the Nervous System Generally. The most frequent cause under this heading is insular sclerosis. There is much similarity between certain cases of acute retrobulbar neuritis and insular sclerosis, and also between the latter and tobacco amblyopia. In insular sclerosis optic nerve changes are not infrequent, and also loss of vision without ophthalmoscopic signs, probably due to an actual affection of the nerve. The insular sclerosis attacks the nerve as it attacks other parts of the nervous system in the form of islands of sclerosis, in which the nerve elements may be interfered with or altogether destroyed. The method by which the pallor travels from the seat of the sclerosis down the disk is not explained. It may possibly be due to wasting of the centrifugal fibres, which go from the basal ganglia to the retina.

Optic Neuritis with Dropping of Watery Fluid from the Nostril. A few cases have been observed in which there was a persistent flow of fluid from one nostril, with headache, vomiting, drowsiness or delirium, and amblyopia. Optic neuritis is present at the same time. The fluid analyzed has been found to resemble cerebro-spinal fluid, in some, if not all of the cases, and there is great probability of its escape from the skull through the cribriform plate of the ethmoid. The prognosis is not good and no treatment has hitherto been found of use.

Atrophy of the Optic Nerve. Atrophy of the optic nerve is either a primary disease or is secondary to some previous affection of the optic nerve, like optic neuritis, embolism of the central artery of the retina, or to retinochoroiditis or retinitis pigmentosa. (Fig. 255.)

FIG. 255.



Ophthalmoscopic appearance of disk in atrophy following hemorrhage. (JAEGER.)

Primary optic atrophy, also known as simple or progressive atrophy, is characterized by gradually increasing pallor of the optic nerves without signs of inflammation. The disease is generally bilateral. The small vessels of the nerve disappear, the retinal arteries dwindle, the veins become funnel-shaped at the disk, and the disk assumes a delicate blue-white tint. The vessels make a slight bend as they pass over the edge of the disk, owing to a slight atrophic excavation of the disk; the stippling of the lamina cribrosa becomes marked, the sight undergoes progressive diminution, perception of color is lost, and the fields become contracted, generally concentrically, but perhaps more in one part than in another. The most common cause of primary optic atrophy is tabes dorsalis. The atrophy is often the earliest sign of tabes and may precede any other symptom by years. Generally where the spinal symptoms come on early, the optic atrophy is late in appearance. Among the other signs are smallness of the pupils, spinal myosis, the Argyll-Robertson

pupil, loss of the knee-jerk, and loss of equilibrium (*Romberg's symptom*), the ataxic gait, while the patient may complain of periodic acute attacks of indigestion (gastric crises), lightning pains in the limbs, or girdle pains about the body. The atrophy of the optic nerve may go on to complete blindness without the appearance of any of the other symptoms, the patient remaining in good health. In some of the cases the disease comes to an end, the optic nerves only are affected; probably these should be regarded as cases of arrested tabes, but generally the signs of ataxy develop later. In complete tabetic atrophy the retinal ganglion cells have been found to disappear; Ward-Holden discovered only degenerated ganglion cells here and there, even in the macula. If this be so, the atrophy of the nerve itself is probably secondary to that of the retinal ganglion cells. Optic atrophy also occurs in insular sclerosis and general paralysis of the insane, and it has in a few rare cases been found associated with diabetes insipidus. It may follow local changes in the orbit, as the pressure of a tumor. These forms are characterized by their being one-sided and affecting only one portion of the nerve. Simple atrophy is rare in children; it is most common after middle age. It is often difficult to distinguish primary atrophy from the atrophy which accompanies very chronic glaucoma. The difference can generally be made out by the fact that in the latter disease perception of colors is not lost, and the ophthalmoscopic appearances show that the excavation of the disk is very much greater in glaucoma than in primary optic atrophy.

The **prognosis** is generally unfavorable; primary atrophy leads almost certainly to blindness.

Treatment should be directed to the disease underlying the atrophy; for the optic nerve itself we may give strychnine or iodide of potassium.

Post-neuritic atrophy is the last stage of an optic neuritis. It differs in ophthalmoscopic appearances from primary atrophy in the appearance of the disk and parts around. Instead of being quite clear and sharply defined, the disk is covered by a light haze, as if it had been washed with Chinese white, the veins are still distended and tortuous, and both arteries and veins have white lines along them, owing to thickening of their sheaths; the lamina cribrosa is hidden by the remains of the inflammatory exudation and there is a disturbance of the choroid around the disk. In many cases, however, it is impossible to say with confidence whether the atrophy is primary or post-neuritic from the ophthalmoscopic appearances alone.

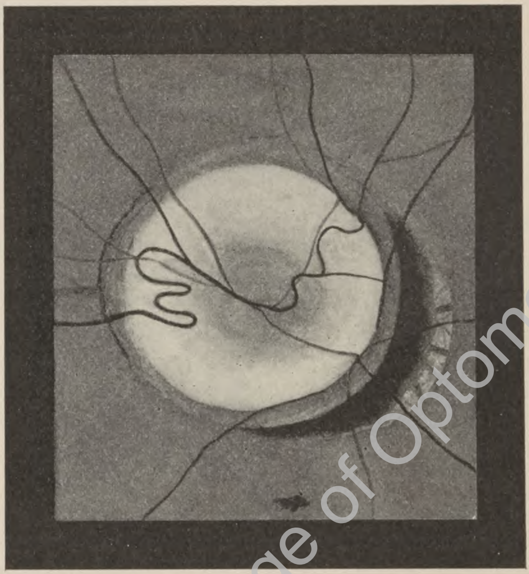
Post-embolic and Post-thrombotic Atrophy. These are distinguished by the obliteration of the affected vessels, which may have shrunk to white bands only or may contain a small visible column of blood, and by a certain amount of change usually seen at the yellow spot. The nature of post-embolic atrophy can generally be determined by the history or by the mode of onset of the affection, that is by its absolute suddenness.

Post-retinitic Atrophy, Waxy Atrophy, Post-choroiditic Atrophy. The papilla in this disease appears to have a dirty yellowish-red color, the vessels are narrowed, and there are signs in the fundus of old choroidal or retinal disease.

In glaucomatous atrophy, typical glaucomatous excavation of the disk, where the vessels disappear at the margin of the disk and are displaced toward the nasal side, is the distinguishing feature.

Tumors of the Optic Nerve. Tumor of the optic nerve occurs rarely. In vol. xix. of the *Transactions of the Ophthalmological Society of the United Kingdom,* Buller and Marshall found only 130 cases recorded in literature. The greater number of cases occurred

FIG. 256.



Coloboma of optic nerve.

before the age of ten years, and the liability to the disease diminished with age. The symptoms of the affection are rapid loss of sight, forward and outward protrusion of the eyeball in the line of the axis of the orbit, little or no limitation of movement, and no pain. (Fig. 256.) The tumor may be felt in some cases behind the eye. The seat of the growth is mostly in the central portion of the optic nerve, and it is not common for the eye to be involved. The kinds of growth found after removal have been gliomata or tumors of the neuroglia, sarcomata, myxosarcomata or endotheliomata. Tuberculous tumors have also been seen.

Treatment. 1. Removal of the tumor with preservation of the eye. This may be done by dividing the external rectus, rotating the eye inward and removing the involved optic nerve, replacing the eye and

suturing the external rectus; the external wall of the orbit may be removed to facilitate access to the tumor (Krönlein's operation).

2. Enucleation of the eye with the involved nerve.
3. Exenteration of the orbit.

Having to deal with a malignant growth, it may be questioned whether it is worth trying to save the useless eye, which is a great hindrance to the easy and efficient removal of the tumor. Having regard to the life of the patient, it is wise to remove the growth as widely as possible.

Injuries to the Optic Nerve. These are caused most frequently by falls on the head. The base of the skull is fractured and the optic nerve is ruptured by splinters of bone, in the optic foramen. Sight may be lost without rupture from hemorrhage into the sheath of the optic nerve. The optic nerve is sometimes injured by foreign bodies penetrating the orbit without the eye being injured. The most familiar instance is by the ferule of an umbrella or walking-stick. The optic nerve is sometimes divided by bullet wounds traversing the orbit, frequently in cases of attempted suicide. In some of these cases intra-ocular hemorrhages and ruptures of the choroid have been found, although the track of the bullet was far removed from the back of the eye. After rupture blindness comes on at once; if the papilla appear to be pale immediately after the injury, the nerve has been ruptured in front of the entrance of the central retinal artery. If the rupture be behind this, the pallor of the disk may not come on for three weeks or longer.

Loss of Sight after Severe Hemorrhages may result from optic nerve atrophy; it may come on from surgical or post-partum hemorrhage. It most commonly results from hemorrhage from the intestinal tract; at first there may be œdema of the retina followed by atrophy of the nerve. If loss of sight be coming on, the patient should be placed in a horizontal position and if possible intravenous injections of saline solution should be made without delay. Sometimes the loss of sight comes on several days after the hemorrhage. The cause of this is not clear, but it is thought by Leber to be due to hemorrhage into the sheath of the nerve creeping forward from the base of the skull. In some cases of injury the blood travels forward and may be seen after several days beneath the conjunctiva and even in the lids. Treatment should be rest horizontally, iron, proper feeding, etc.

Hyaline Growths in the Optic Papilla. Hyaline nodules growing from the lamina vitrea of the choroid are very common. In rare cases they appear on the disk itself; they are gray nodules clustered together, generally at the edge of the disk. They do not as a rule interfere with vision, and no treatment is called for.

Ocular Signs and Symptoms Attending Diseases of the Brain.

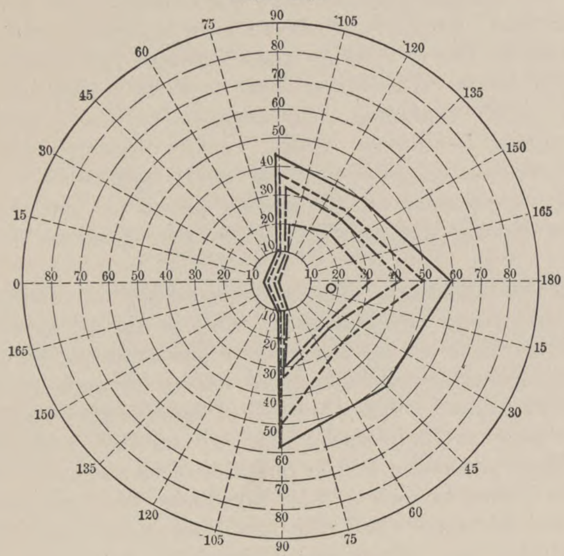
Optic neuritis and loss of sight may indicate tumor of the brain; they are of value as showing the presence of a tumor only; they have

no localization value. The form of optic neuritis known as choked disk is the one which commonly attends cerebral tumor, but the neuritis may also be combined with retinitis without much swelling, and may resemble albuminuric retinitis, even when the cause is an intracranial growth. In case of doubt, examination of the urine should be made. In meningitis and hydrocephalus optic neuritis may also be present. Cysts and hemorrhages, as a rule, do not give rise to ocular symptoms, but an abscess of the brain may do so. Optic neuritis in cerebral tumor occurs early, but it may be delayed or it may not occur at all. The sight may be unaffected for a long time, but the field of vision soon becomes somewhat contracted. In other cases in which the neuritis is axial, loss of vision in the form of a central scotoma may occur early. Optic neuritis may be caused by a tumor of any size in any part of the brain. Tumors of the cerebellum and at the base of the brain are more likely to produce optic neuritis than tumors in other parts, probably owing to pressure on the veins of Galen. The next most important ocular sign in disease of the brain is hemianopsia or loss of half of the field of vision. This is due to a disease of the chiasm, of the optic tracts, of the cortical centre of vision, or of some part of the path connecting the optic tracts with the cortex of the brain. Local disease of the eye producing loss of half the visual field is not included under the name of hemianopsia. When hemianopsia occurs, the fields are usually both affected, one-half of each field being normal, the other half being blind. The dividing line between the two is a vertical one through the centre of the fields, but usually turning aside a little at the fixation point in each eye so as to leave it unaffected. In some cases, however, it goes through the fixation point. It sometimes happens also that the line of separation is not quite vertical, but somewhat irregular, so that the seeing half of the retina passes over the middle line. The blind part is generally absolutely blind, but in some cases color perception only is lost, so that we have a condition of hemianopsia for colors or hemiachromatopsia. In some cases the defect is sectorial, and in other rare cases it is the upper or lower halves that are defective. The most frequent form is one in which the corresponding halves in each eye are wanting, for instance, the right half of each field may be blind, implying the loss of function in the left half of each retina, or if the left half of the field be blind the right half of each retina will be functionless. This is called homonymous hemianopsia. (Figs. 257 and 258.) Many cases of double homonymous hemianopsia have been described due to disease of the cortical visual centres in each hemisphere. Most of the cases have begun with loss of vision in corresponding halves of each eye, followed at a later date by loss of vision in the other halves, whereby total blindness was produced. In a few of the cases the macula was left, so that there was fairly good vision while looking ahead, but the patients had no power of directing their movements owing to the small size of the visual field (loss of orientation). Loss of the outer halves of each visual field or loss of function of the two

nasal halves of the retina is known as bitemporal hemianopsia. Nasal hemianopsia is blindness of the inner half of each visual field, and is due to a want of function on the temporal side of each retina. To

FIG. 257.

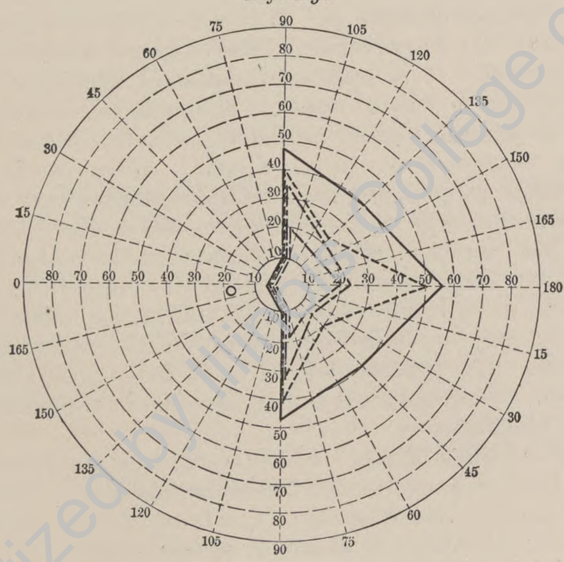
Right Eye



Left-sided hemianopsia.

FIG. 258.

Left Eye



Left-sided hemianopsia.

explain the cause of the hemianopsia in these affections, it will be necessary to go over afresh the course of the visual fibres from the retina to the cortex of the brain.

The nerve fibres from the corresponding or homonymous halves of each retina—that is, from the nasal half of the right and the temporal half of the left retina—pass through the chiasma and unite to form the left optic tract; similarly fibres from the temporal half of the right and nasal half of the left retina pass through the chiasma to form the right optic tract. The fibres from each tract pass into the basal ganglia, the optic thalamus, the anterior corpus quadrigeminum, and the external geniculate body; thence through the posterior part of the internal capsule, and the optic radiations to the visual centre. This is situated on the mesial surface of the occipital lobe in the region having the middle part of the calcarine fissure at its centre.

Lesions of the Chiasma. The most usual sign of affections of the chiasma is bitemporal hemianopsia. This is due to involvement of the decussating fibres at the anterior or posterior border of the chiasma. These fibres supply the nasal halves of each retina, which are consequently blind. The affection of sight begins by a limitation of the outer part of each visual field, or one-half may be affected before the other, according to the position of the lesion. The loss of sight may for a time be only a color blindness, but later on light and form sense are involved also; the loss goes on to total bitemporal hemianopsia. In many of the cases, owing to advance of the disease, the uncrossed fibres become affected too, and the result is total blindness. It has been demonstrated by Nettleship and others that chiasmal disease frequently begins as a central scotoma, and as such may be mistaken for toxic amblyopia. It is believed in such cases that the disease starts in the anterior part of the chiasma, where the macular fibres are situated. It is common in most of the cases to have a certain diminution in central vision. The optic nerve becomes atrophic, there is generally great headache, sometimes loss of mental power; optic neuritis is not often present. Affections of the outer side of the chiasma producing nasal hemianopsia are very rare; horizontal hemianopsia may be caused by pressure on the chiasma above or below. The causes of chiasmal disease are acromegaly, tumors of the pituitary body, meningitis, frequently syphilitic, periostitis of the body of the sphenoid, tubercular masses, syphilitic gummata, cysts and exostoses, and hydrocephalus produced by distention of the third ventricle. The treatment must be in accordance with the discovered cause. Much good may often be done in syphilitic cases by appropriate treatment.

Lesions of the Optic Tract. The characteristic symptom of lesion of the optic tract is homonymous lateral hemianopsia. This may be complete or partial, involving only a quadrant of each retina, as in a case related by Henschen, in which there was a defect of the field of vision in both left lower quadrants, caused by a tumor pressing on the

upper part of the right optic tract. The defect may be relative also—that is, there may be half-vision for color only—form and light perception being unaffected. Owing to proximity of the trunks of the nerves at the base of the brain, there may be paralysis of the parts supplied by these nerves owing to pressure from a tumor of the tract. Optic neuritis may be present in localized meningitis or in tumor, and primary optic atrophy is sometimes met with; both these manifestations are often more marked in one eye than in the other. Homonymous lateral hemianopsia is shown by blindness of the corresponding halves of the retina, for instance, the temporal half of the left retina and the nasal half of the right. Wernicke's sign or the hemiopic pupil is frequently present, and is of great value in localizing the seat of the affection; it consists in the fact that light thrown upon the blind half of each eye produces no pupil reaction at all, while light thrown upon the acting half of each retina gives rise to normal pupil reaction. Afferent impulses proceeding centrally from the retina are interrupted at the seat of the disease in the tract, and do not pass to the basal ganglia and third nerve nucleus, while lesions of the visual path above the basal ganglia producing homonymous hemianopsia do not interfere with the path of the impulses, which pass from the retina to the basal ganglia and thence to the third nerve nucleus. It is conceivable, therefore, that there might be symmetrical lesions in the hemisphere, which produced double homonymous hemianopsia, and therefore total blindness of each eye, and which yet left the pupillary light reflex unaffected. The hemianopic pupil reaction is not easy to obtain, inasmuch as it is difficult to keep the light passing to one side of the retina from illuminating the other half to a certain extent. But it is generally possible to establish a difference between the reflex action of the pupil, when light is cast from opposite sides on to the retina in diseases of the tract. (For the method of applying the test, see page 34.) Symptoms of disease of the optic tract may be produced by the pressure of tumors of neighboring parts, of the temporosphenoidal lobe, optic thalamus, or crus cerebri.

Lesions of the Basal Ganglia or Primary Optic Ganglia, External Geniculate Body, Optic Thalamus, and Anterior Corpus Quadrigeminum. These ganglia undergo degeneration after removal of the eye; also after lesion of the occipital lobe, degeneration can be traced down to them; they are therefore in the direct line of transmission of impulses from the eye to the brain cortex.

External Geniculate Body. All the fibres of the optic tract enter the external geniculate body, and a lesion here always gives rise to hemianopsia. It is probable also that its upper and lower parts supply the upper and lower parts of the retina, respectively. If so, homonymous quadrant hemianopsia may be due to disease of the external geniculate body.

Optic Thalamus. Lesions of the posterior part of the optic thalamus, the pulvinar, have in many cases been found in association with

hemianopsia, but in an equal number of cases hemianopsia has not been found. It is probable that the lesion of the optic thalamus itself has not given rise to hemianopsia except indirectly from pressure on the optic tract.

Corpora Quadrigemina. It is not certain that tumors of the corpora quadrigemina give rise to loss of sight. A few doubtful cases have been recorded in which there was blindness from lesions in this situation, but there is a far larger number of cases in which tumors of the corpora quadrigemina produced no blindness at all. (For oculomotor affections following lesions of the corpora quadrigemina, see page 180.)

Lesions of the Internal Capsule. The course of the visual fibres from the external geniculate body to the optic radiation is not yet known. Fibres are traced to the external geniculate body through the posterior third of the posterior limb of the internal capsule, and it is asserted that a lesion of this part of the internal capsule causes hemianopsia. On the other hand, Henschen has shown that lesions of this part do not necessarily cause hemianopsia, and that when they are associated with it, the external geniculate body or the optic tract is interfered with at the same time.

Lesions of the Optic Radiations. It is unknown with accuracy how large a portion of the optic radiations is occupied by the visual fibres. According to Henschen, only the central portion is so occupied. Other writers agree with him that the optic radiations contain many other fibres than visual ones. The importance of this lies in the fact that in a lesion of the optic radiation, although we may get hemianopsia, we get also other symptoms, which may aid us to recognize the lesion and to distinguish it from a cortical one. A subjective sensation of blindness is caused by a lesion of the radiations, in the form of positive scotoma, but it is not present in a lesion of the cortex. If the lesion extends beyond the visual fibres, various other symptoms are present, such as mind blindness, word blindness or alexia, visual aphasia, dyslexia, amnesic color blindness, or visual hallucinations.

Alexia or Word Blindness. In this affection persons are unable to read words; the print or writing is perfectly well seen, the letters themselves, except in rare cases, can be made out, but the power of combining them is entirely lost. The patient can write quite well, but is unable to read anything he has written unless he is allowed to go over it with a pen. In some cases even individual letters cannot be recognized, but figures can as a rule be read. Word blindness may be combined with an inability to write.

Alexia with Agraphia. This alexia is due to interference with the fibres passing from the visual memory to the speech centre, and has a distinct localizing value as the lesion has been recorded from post-mortem examination in five cases in the left occipital lobe. Alexia with agraphia is thought to be due to a lesion of the centre for visual memory in the left angular gyrus. The association of alexia with right homonymous hemianopsia is to be explained by the

nearness of the visual centre and paths to the lesion which cause alexia.

Dyslexia. In this affection there is no loss of sight, but a simple inability of the patient to read continuously. A few words only can be read, then the book is thrown aside; the effort may be repeated after a time, but power of continued reading is absent. In most cases dyslexia has been associated with hemianopsia and other cerebral symptoms. It is caused by degeneration of the cerebral vessels and generally has a fatal termination; it appears to have little value as a localizing sign.

Visual Aphasia. The patient cannot remember the names of things seen, although quite familiar with the things themselves, but can remember their names if he can recognize the things by some other sense than that of sight, as, for instance, if he can touch them. Conversation of patients suffering from visual aphasia has certain well-marked peculiarities—the general avoidance of names and the use of circuitous methods of speech, in order to make up for the defective power of expression. Right homonymous hemianopsia is almost always present and sometimes alexia and agraphia. The lesion is believed to be in the left occipital lobe.

Loss of Color Memory—Amnesic Color Blindness. The patient is able to perceive the colors and to match them correctly, but is unable to give them their names. This has always been found associated with right homonymous hemianopsia, and is believed by Wilbrandt, who described it, to be an indication of disease of the occipital lobe on the left side, preventing communication between the color centre for vision and the speech centre.

Visual hallucinations sometimes occur in the blind side of the field; objects are seen in the blind area which are not in view at all. This is thought to be due to irritation of the centre for visual memory in the occipital lobe, but a case has been published by de Schweinitz in which hallucinations occurred in the blind side of the field, due to a gumma pressing on the right optic tract. Visual hallucinations are always accompanied by right homonymous hemianopsia. Hallucinations of vision due to focal brain disease are generally associated with other signs of focal brain disease, and may be distinguished from other visual hallucinations, such as those occurring in delirium tremens or fever, by their occurring in the blind part of the visual field.

Mind Blindness or Visual Amnesia. The sight is perfectly good, intelligence is not affected, the patient is able to read, but he has lost the power of recognizing objects seen; if they be presented to him by one of his other senses, he at once recognizes them People, even most intimate friends, are not recognized by their appearance, but are known at once when they begin to speak. This failure is due to a lesion of the centre for visual memory, which is supposed to be distinct from the visual centre, although situated close to it in the occipital lobe. It serves for the storing up of pictures in the memory of objects or scenes that have fallen upon the retina, and whenever a picture

is formed on the retina the stores of the visual memory centre are brought out and ransacked for comparisons or the new picture is stored away for future use. Homonymous hemianopsia is present in the majority of cases of mind blindness. It occurs in persons beyond middle age, when the lesion has been found to be hemorrhages, softenings, or tumors; it occurs also in general paralysis of the insane.

Lesions Affecting the Cortical Centre of Vision. According to the most recent researches of Henschen, th s lesion is placed about the middle part of the calcarine fissure, the upper edge of the fissure representing the upper homonymous quadrants of the retina and the lower edge of the fissure, the lower homonymous quadrants, the macula centre lying in the floor of the fissure. Other observers think the visual centre extends much more widely, even covering the whole mesial surface of the occipital lobe, but they agree in giving special importance to the calcarine fissure. Destruction or lesion of the cortical visual centre leads to absolute blindness of the corresponding halves of each retina, and consequently to homonymous hemianopsia of the opposite half of the field of vision. If the lesion be confined to the cortex, there is complete absence of any other sign, such as paralysis, anæsthesia, word blindness, mind blindness, visual aphasia, visual hallucinations, and the hemianopic pupil. For instance, there is complete absence of sensation in the blind side of the field, but the patient is not conscious of the defect as of a dark area, as he may be in lesions of other parts of the visual path. If the lesion be bilateral, of which many cases are on record, the symptoms are bilateral homonymous hemianopsia, which means complete loss of vision. In small lesions less than half the homonymous fields may be lost and partial peripheral or more rarely scotomatous defects may be left. But these partial homonymous defects should be used with caution for localizing purposes, as homonymous peripheral contractions of the fields of vision are found in patients without lesion of any part of the visual path. Sometimes the blindness is incomplete—that is, perception of light may be retained in the blind part of the field In other slighter cases again only the color sense in that half of the field may be lost (hemiachromatopsia), light and form sense being perfect Various degrees of loss may be present in different parts of the affected fields. It is unknown whether the centre for colors is different from those for form and light, or whether the different colors have separate cells devoted to them; but there are cases on record of homonymous hemianopic losses of perception of one color which would give support to this view. Probably there are not separate centres for light, form, and color, but a loss of the latter indicates a less serious injury to the centre than when its other functions are lost. It has been stated that in most cases the dividing line between the two halves of the fields is not a perfectly vertical one, but that it deviates so as to include the whole of the fixation point in each seeing half of the field. Two explanations may be given of this: (1) That the whole of the macular region of each eye is represented in the visual centre of each side, so

that each macula has a double nerve supply, and if one visual centre is destroyed, it still retains its nerve supply to the visual centre of the opposite side. (2) The other explanation given for retention of the whole central vision in lesion of one visual centre is that the centre for the macula in the cortex is more vascular than the rest of the visual centre, and receives nutrition from anastomoses even when parts around are cut off.

It has been supposed by some writers that there is a higher visual centre in the angular gyrus in which the whole of the opposite field of vision is represented, that of the same side being also represented, but in a lesser degree of intensity. Lesion of this centre is said to cause amblyopia of the opposite eye by lowering the function of the whole retina and reducing the size of the visual field generally, without producing hemianopsia (*crossed amblyopia*). It also produces a slight constriction of the field of vision of the same side. Ferrier's experiments on animals support the view of the existence of a centre for vision in the angular gyrus, but the evidence in favor of it in man is very slight, and many writers do not believe in the existence of crossed amblyopia.

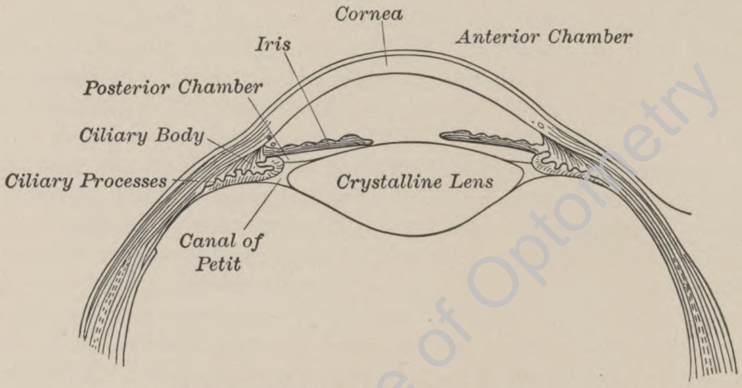
CHAPTER X.

DISEASES OF THE CRYSTALLINE LENS.

By EDWARD C. ELLETT, M.D.

Anatomy. The crystalline lens, or, as it is commonly called, the lens, is a biconvex transparent body which lies in the anterior portion of the eye, and, together with its suspensory ligament, serves to separate the vitreous chamber behind from the aqueous chambers in front. (Fig. 259.) The posterior surface is the more convex.

FIG. 259.

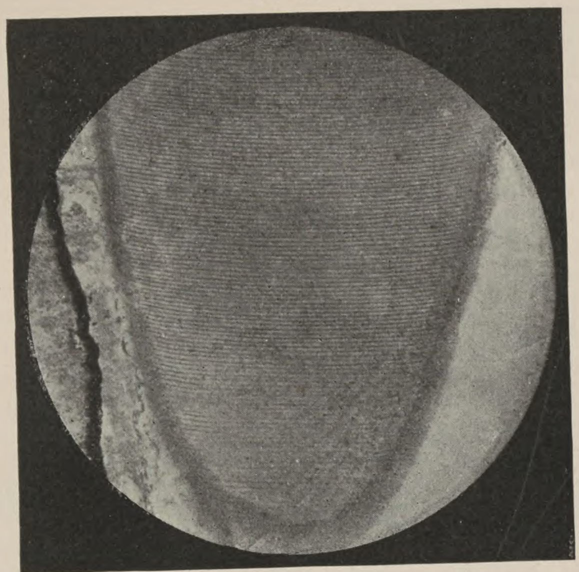


The anterior segment of the eye. (Modified from Gray's Anatomy.)

The lens is composed of a harder central portion known as the nucleus, and a softer outer portion known as the cortex. The boundary line between these two portions is not sharply defined, the nucleus deriving its greater density from a process which consists essentially in a loss of fluid, and this being a progressive process the nucleus increases in size at the expense of the cortex in proportion to the age of the individual. The nucleus has a yellowish color as compared to the cortex, and also a greater refractive power. In elderly persons these properties frequently make the lens as seen through the pupil present a grayish or opaque appearance, due to increased reflection of light from the surface of the lens (increased lens reflex), and this may be mistaken for opacity of the lens, although the vision is not impaired by it, and examination with reflected light (ophthalmoscope) shows the lens to be perfectly clear.

The lens is composed of prismatic fibres joined together by a small amount of cement substance. (Fig. 260.) These prismatic fibres

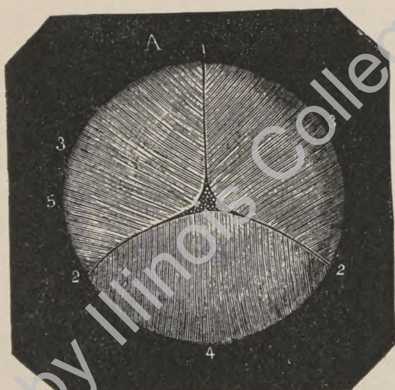
FIG. 260.



Normal lens fibres cut longitudinally. × 100. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

are elongated epithelial cells, and are arranged in bundles, these bundles being so placed that their lines of union form a stellate figure

FIG. 261.



Sectors in crystalline lens. (TESTUT.)

radiating from the centre of each surface of the lens, and often visible on oblique illumination in the normal eye. (Fig. 261.) Besides

the nucleus and cortex, we distinguish the poles of the lens; that is, the centre of its anterior and posterior surfaces, known respectively as the anterior and the posterior poles. The circumference of the lens is known as the equator.

The average size of the lens is 9 mm. in diameter and 4 mm. in thickness at the central or thickest part. From the centre it slopes away gradually until the two surfaces meet at the thin edge or equator.

The lens is enclosed in a delicate structureless lining membrane of perfect transparency, known as the capsule. This is divided into two portions: that on the anterior face being called the anterior capsule, that on the posterior, the posterior capsule. The anterior capsule is lined on its lenticular surface with a layer of epithelial cells, from which new lens fibres are developed.

The lens rests in a cup-shaped cavity of the vitreous, called the fossa patellaris, or hyaloid fossa. In front it is separated from the pupillary border of the iris by a thin layer of the aqueous humor, this layer being so thin that the pupillary border of the iris and the anterior lens capsule may be considered as being in contact. The lens is held in place by its suspensory ligament, called the zone of Zinn. This is a fibrous structure which arises from the pars ciliaris retinæ as far back as the ora serrata, the surface of the ciliary body, and the ciliary processes. It leaves the wall of the eye at the ciliary processes, and divides into two layers, an anterior and a posterior, which unite with the anterior and posterior portions, respectively, of the lens capsule. The space between these two layers of the suspensory ligament is known as the circumlental space, or canal of Petit, and is more or less completely divided into two parts by a delicate septum running from the ciliary processes to the equator of the lens. The anterior layer of the suspensory ligament presents numerous small openings whereby the canal of Petit communicates with the posterior chamber, both being lymph spaces and both containing aqueous humor. (Fig. 259.)

The lens is an elastic body capable under certain conditions of changing its shape, as is described in the account of the function of accommodation.

The lens does not contain any bloodvessels, but derives its nutrition by imbibition from the fluids surrounding it. The absence of bloodvessels prevents it from presenting phenomena of inflammation, and the way in which it is nourished explains why inflammatory conditions of the choroid exert a deleterious influence on it.

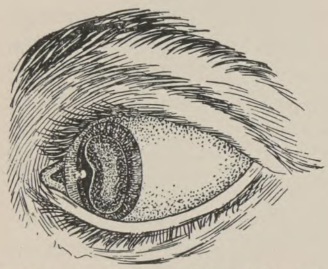
Embryology. Very early in the development of the embryo the outer layer, or epiblast, is thrown into a longitudinal dorsal furrow whose sides close over to form a tube, the medullary tube. From the anterior end of this tube are thrown out processes on each side, called the primary optic vesicles. Each of these vesicles is converted into a cup by the anterior wall receding against the posterior wall. The layer of epiblast over this cup-shaped cavity thickens, dips into the cup, and this portion gradually becomes cut off from

the rest of the epiblast and forms an isolated mass of epiblastic tissue lying in the cup of the optic vesicle. From this mass of tissue the lens develops. This mass of epiblastic cells is composed roughly of two layers of cells, an anterior and a posterior. From the posterior layer, by a process of elongation, the lens fibres are formed, the anterior layer of cells remaining as a thin layer of cells just under the anterior capsule. From the most equatorially situated of these cells additional lens fibres develop. The lens attains its full growth about the eighteenth year, after which time the formation of new fibres continues much more slowly, at a rate to compensate for the diminution in size of the central portion from contraction.

It will be seen that at one time the rudimentary lens occupies nearly the whole of the rudimentary eyeball. At the time of birth, however, the lens has become reduced to almost its normal relative size, although, as stated, further changes occur until the eighteenth year.

The lens capsule is developed from a layer of mesoblastic tissue which surrounds the plug of epiblastic tissue when it grows into the optic cup to form the lens.

FIG. 262.



Kidney-shaped lens, coloboma inward. (BAAS.)

FIG. 263.

Lenticonus anterior. (WEBSTER.)

FIG. 264.

Dislocation of the lens.

Congenital Anomalies. Congenital anomalies of the lens may affect its size, shape, position, and transparency.

Anomalies of Size. Congenital absence of the lens (aphakia) has been noted a few times. It results either from arrest of development or from disease.

The lens varies in size at different ages. The only anomaly of size usually recognized is that in which the lens is too small (microphakia). There is often a relaxation or absence of the suspensory ligament at the same time, which permits the lens to change its position.

Anomalies of shape are of two sorts: (1) coloboma lentis, and (2) lenticonus.

Coloboma of the lens is the name applied to a condition in which the edge of the lens presents at some point, usually downward, a localized flattening or a distinct notch. (Fig. 262.) It is due to an arrest of development. This appearance varies in shape and position, and while usually single, the edge of the lens may be notched

in several places, or serrated. This anomaly is often associated with coloboma of the iris and choroid.

Lenticonus is a condition in which a more or less pointed projection exists on the anterior or posterior surface of the lens. (Fig. 263.) The projection is transparent as a rule, but an opacity may exist at its apex. The cause is obscure.

Anomalies of position are grouped under the name of ectopia lentis or displacements of the lens. Except in the rare cases where there is an arrest of development of the whole organ and the lens remains in its fœtal position in the vitreous chamber, the dislocation occurs in the direction of the equator, and is due to faulty development and consequent weakness of some part of the suspensory ligament. This weakness generally exists in the lower part of the eye in the position of the fœtal cleft, and the dislocation is in the opposite direction; that is, upward and inward or upward and outward. (Fig. 264.) The whole ligament may be absent, permitting the lens to pass through the pupil and lie in the anterior chamber. This condition is usually bilateral and symmetrical, but may be unilateral. It is also frequently hereditary, when the anomaly is found in successive generations of a family. The displacement varies much in degree, so that the patient may see either through the lens or through the part of the pupil which contains no lens, or, according to the position of the head, either through the lens or unobstructed pupil at will. The lens is at first clear, and remains so in the majority of cases, but may become opaque in time. Sometimes it is fixed in its abnormal position, and sometimes freely movable, depending on the condition of the suspensory ligament.

The **treatment** of ectopia lentis may often be satisfactorily conducted by means of correcting lenses. If the dislocated lens is so situated as to allow vision always through that body, or always through the unobstructed pupil, glasses to correct the refraction, concave as a rule in the former case and strongly convex in the latter (just as in aphakia), will often give useful vision. In other cases it will be impossible to secure satisfactory vision in this way, and the lens must be removed by the operation of solution, to be described later. This treatment results in absorption of the lens, leaving the eye aphakic, and requiring strong convex glasses, as will be mentioned in considering the treatment of cataract. Where the lens becomes opaque, it is usually advisable to remove it by the same means.

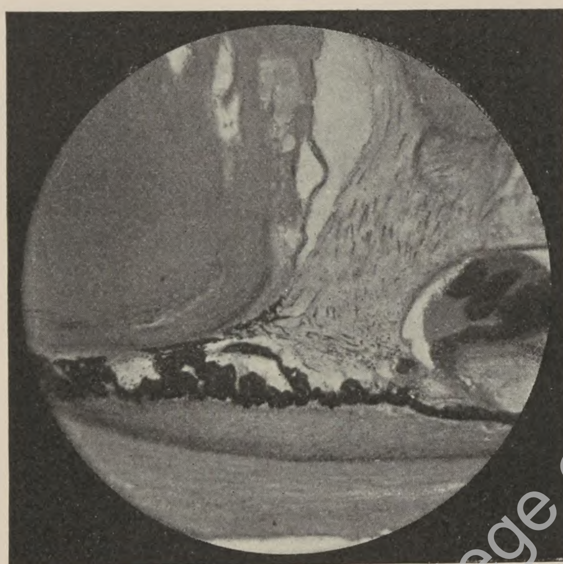
Anomalies of transparency include the various forms of congenital cataract. These are: 1. Anterior polar cataract. 2. Posterior polar cataract. 3. Lamellar or zonular cataract. A detailed description of these and the method of treating them will be given in considering the subject of opacities of the lens.

Wounds and Injuries. Injuries to the lens are of two kinds: First, the lens is displaced from its normal position, constituting traumatic luxation of the lens. Second, the lens is the seat of a penetrating wound which is usually followed by traumatic cataract.

Traumatic displacements of the lens are the result of injuries to the suspensory ligament. The displacement may be partial or complete.

Partial displacements of the lens (subluxation) result from an injury whose effect is so localized as to rupture only a portion of the suspensory ligament, and without leaving its normal position the edge of the lens nearest the seat of rupture tilts forward, causing astigmatism and consequent disturbance of vision. The lens, as a rule in these cases, remains transparent, and the treatment consists in correcting the resulting astigmatism by glasses. Considerable spontaneous improvement may occur, or the lens may become opaque, when it is dealt with as if the opacity resulted from other than trau-

FIG. 265.



Lens luxated upon the ciliary body. The lens is becoming cataractous and is bound down by fibrous tissue. From an eye which caused sympathetic irritation in the fellow eye. × 15. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

matic causes. (Fig. 265.) The lens may in its new position cause so much inflammatory disturbance as to result in the loss of the eye.

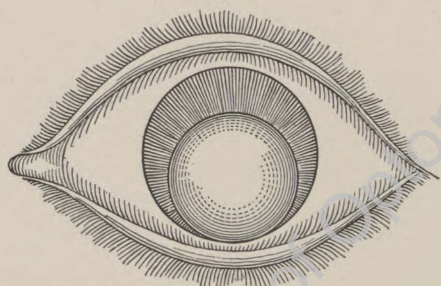
Complete dislocation of the lens follows an injury which causes complete rupture of the suspensory ligament, thus permitting the lens to leave its bed entirely. The capsule of the lens is generally not ruptured. The dislocation may occur in one of several directions: 1. Forward into the anterior chamber. 2. Backward into the vitreous chamber. 3. Through a rupture in the coats of the eyeball into the capsule of Tenon or under the conjunctiva. Owing to the elasticity of the latter membrane, it will sometimes not yield to an injury which ruptures the sclerotic, choroid, and retina, and the lens

passing through a rupture in these coats lodges under the conjunctiva, which stretches to accommodate it.

The symptoms of dislocation of the lens are, in the first place, dimness of vision. By removal of the lens from an eye whose refraction is hyperopic or moderately myopic, rays of light are no longer focused on or sufficiently near the retina to permit of clear vision. Other conditions which result from the injury may also contribute to cause dimness of vision, such as vitreous hemorrhages, rupture of the choroid, hemorrhage into the anterior chamber, etc. There are two conditions in which dislocation of the lens would cause improvement instead of deterioration of vision. These are when the lens is opaque and when the eye is highly myopic. In both of these conditions operations for the removal of the lens are resorted to for the purpose of improving vision, by removing a mechanical obstruction to sight in the first instance, and by changing the refraction of the eye in the second instance.

If the lens is dislocated into the anterior chamber, it can be seen as a clear or opaque disk, as the case may be, in this position. (Fig. 266.) If clear, the iris and pupil can be seen through it. It is apt

Fig. 266.



Opaque lens dislocated into the anterior chamber.

to excite inflammation in the eye, and invariably does so in that part of the cornea with which it is in contact. It may also cause glaucoma. It should be removed from the eye by solution or extraction.

If dislocated into the capsule of Tenon, which is very rare, or under the conjunctiva, the lens can be seen in its new position, and its absence from its normal position is indicated by the fact that the refraction of the eye is highly hyperopic and the catoptric images cannot be seen. If not itself disturbed by the injury, the iris is seen to be tremulous on movement of the eye, because it no longer has its normal support behind. The gravity of this condition does not pertain to the lens or its new position, but to the rupture of the coats of the eye, and varies with the site and extent of this rupture.

The lens may be left alone. If it is deemed advisable for any reason to extract it, this should not be attempted until the scleral

wound has healed, as we would otherwise convert a simple scleral wound, to borrow an analogy from general surgery, into a compound one, and through this, protrusion of the other coats or ocular contents may occur, adding greatly to the gravity of the injury. When the scleral wound has healed, the lens may be removed through an incision through the conjunctiva (and capsule of Tenon when necessary), placed as far as possible from the scleral scar.

Dislocation into the vitreous chamber is by far the most common form of traumatic dislocation. The lens loosened from its attachments sinks downward and backward into the vitreous, where it may be made to rise and be seen through the pupil by up-and-down movements of the ball similar to those practised for the study of opacities in the vitreous (ocular ballottement). It was formerly the custom to treat cataract by depressing the lens downward and backward into the vitreous, and we have abundant records of the effect of this form of dislocation. Sooner or later the lens, acting as a foreign body, will excite inflammatory and degenerative changes in the vitreous, choroid, or retina, which destroy or greatly impair the sight. It may also cause glaucoma. For these reasons this method of treating cataract has been abandoned, and for these reasons it is desirable to remove the lens from the vitreous if possible. The operation is dangerous on account of the tendency of the vitreous to escape from the eye when an incision is made into the cornea. The lens, too, is far from the corneal incision, and must usually be brought forward by means of a delicate wire loop. To facilitate the capture of the lens, it has been suggested to operate with the patient lying face downward, this favoring the seeking by the lens of its natural position, or the lens may be coaxed into this position and fixed there by a two-pronged needle or bident thrust into the eye through the sclerotic behind the ciliary process, and thus behind the lens, which it holds in place by pressure. The steps in the removal of the lens will be sufficiently detailed in speaking of the treatment of cataract.

In whatever position the lens may be dislocated, it almost invariably becomes opaque sooner or later.

Wounds of the Lens and Traumatic Cataract. It is very nearly true that a wound of the lens means traumatic cataract; but traumatic cataract may be due to other injuries than wounds of the lens. In other words, traumatic cataract may occur without rupture of the lens capsule. This is not common, but it has been observed that contusions and concussions of the eye may be followed either immediately, or after some days or weeks, by the formation of opacities in the lens. These opacities may be stationary or progressive, although there is very seldom any clearing up of them observed, and it is certainly not to be expected. This form of lenticular opacity is more apt than any other to remain stationary, or if it progresses it does so at a slow rate. The prognosis to the eye from this form of cataract, *per se*, is therefore good. Progressive opacities of this

character require removal of the lens by operation, either by extraction or by solution. The choice between these methods will, as a rule, depend on the consistency of the lens as estimated by the age of the patient, solution being adapted to patients under twenty-five or thirty years, and extraction to patients over that age.

We take advantage of the possibility of causing opacity of the lens by contusion without rupture of the capsule in the operation of massage of the lens, which is sometimes performed to hasten the maturation of a cataract, to be described later.

Traumatic cataract from rupture of the capsule of the lens usually results from a penetrating wound. The formation of this variety of traumatic cataract depends on the fact that if the aqueous humor comes in contact with the fibres of the crystalline lens it causes them to swell and become opaque, and in time completely dissolves the lens after it has undergone the process of swelling and opacification.

FIG. 267.

Traumatic cataract, showing splitting up of fibres and formation of spherical masses. There are numerous swollen fibres which show transverse striations. × 100. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

(Fig. 267.) Rupture of the capsule and traumatic cataract may result from a contusion of the eye, but for the purpose of study the process is observed best where it follows the operation of needling the lens. Here a wound is made in the capsule and lens with a needle thrust through the cornea. By oblique illumination and a magnifying lens the rent in the capsule can be seen at once; but the track of the wound in the substance of the lens is not usually visible for several hours. As the aqueous gains access to the lens through the rent in the capsule, the portions nearest the opening become opaque and swollen, and are apt to be extruded into the anterior

chamber, as there is not room for them in their swollen condition within the capsule. We can thus find one or more masses of opaque lens matter lying in the anterior chamber. The process continues, more and more of the lens breaking down and being pushed out into the anterior chamber. Sometimes when the wound of the lens is small the aqueous humor filters in along the wound path, and finds its way from this along the interspaces between the lens fibres, showing as beautiful festoons of opaque lines, sometimes resembling the skein figures seen in the process of karyokinesis.

In the process as described, the absorption of the lens is supposed to advance in a gradual way to its completion. It very often happens that the wound in the capsule is so large that a considerable portion of the lens is subjected to the influence of the aqueous, and becomes swollen at once. In this case the augmentation of the intra-ocular contents is so great that the tension is increased and glaucomatous symptoms appear.

Penetrating wounds of the lens are necessarily complicated by a wound of some coat of the eye, generally the cornea, through which the wounding body reaches the lens. Frequently the iris is also wounded, and the offending substance may go through the lens to the deeper parts of the eye. The wound may be so extensive that the injury to the lens becomes of minor importance compared with the serious nature of the injury to other structures.

We have spoken only of traumatic cataract resulting from the action of the aqueous humor. In a few cases it has been observed that the posterior capsule alone was ruptured, and opacity of the lens resulted from the action of the vitreous humor. The action of the vitreous in this respect is very much less intense and rapid than that of the aqueous, but it should be remembered that traumatic cataract may follow a rupture of the posterior capsule.

Where the injury is wrought by a small foreign body, this sometimes lodges in the lens. Its principal effect is the production of cataract, the presence of the foreign body adding very little to the gravity of the case. Even if the foreign body is infected, the lens being peculiarly resistant to infection from pathogenic germs, suppuration in the eye does not necessarily follow.

When the wound of the capsule is small, it may close so quickly and so completely after the wound is made that the lens substance is not exposed to the action of the intra-ocular fluids. This is analogous to the manner in which the corneal wound closes behind the needle in the operation of discission, and the aqueous humor does not escape.

A rare form of traumatic cataract is that caused by heating of the aqueous humor in applying the actual cautery to the cornea in certain diseases of that membrane.

Treatment. The treatment of traumatic cataract consists, in the first place, in an effort to secure mechanical and surgical cleanliness of the wound on the surface of the eye. Any protruding portions

of iris or other of the ocular contents are replaced or excised and the conjunctival sac flushed with a mild antiseptic solution. This part of the treatment is dealt with in detail under the head of Wounds of the Cornea and Iris. Atropine is instilled, and the patient put to bed. The object of the atropine is to place the eye at rest, and, by withdrawing the iris into the periphery of the anterior chamber, to allow room for the swelling of the lens. The solution generally used is of the strength of four grains of sulphate of atropine to the ounce of distilled water, the solution and dropper being sterilized. It is a good plan to incorporate some non-irritating antiseptic, such as boric acid, with the solution; otherwise it is difficult to keep the solution sterile, as the receptacle containing it is frequently opened and the solution exposed to the air. A light antiseptic dressing and bandage are applied, at least until the external wound has closed.

If reaction is excessive, it is controlled best by the application of ice compresses and the internal administration of calomel in doses of one-tenth of a grain, combined with bicarbonate of sodium, every hour until free purgation or "touching of the gums" makes it advisable to discontinue it. As calomel is given in this way for its antiphlogistic effect, and often fails to purge, a saline cathartic had best be also given, and the patient's general health and secretions kept in good condition. Under this treatment traumatic cataract will often be gradually dissolved. Excessive swelling of the lens may occur, producing glaucomatous symptoms. The eye becomes the seat of intense pain which radiates to the frontal, temporal, malar, and even occipital region. Conjunctival injection is marked, and palpation reveals an increased intra-ocular tension. These symptoms are readily explained by finding the anterior chamber filled with the swollen and opaque lens matter. This condition necessitates evacuation of part or all of the lens matter by the operation of simple linear extraction, which will be described in considering the treatment of cataract. It is best to remove all of the lens matter that can be removed, since this not only more effectually relieves the glaucomatous symptoms, but renders their recurrence less probable and hastens the cure of the condition by leaving only a small amount of the lens to be dissolved by the aqueous humor.

Another potent reason for evacuating the swollen lens matter when considerable in amount was mentioned in speaking of dislocation of the lens into the anterior chamber, namely, that pressure of the lens on the posterior surface of the cornea may excite inflammation or even sloughing of that membrane.

In some cases of traumatic cataract, especially where the wound is small, the lens becomes opaque, but is not absorbed. Under these circumstances it should be removed by solution or extraction in the same manner and for the same reasons as if dealing with a monocular cataract due to other than traumatic causes. In children a cataract in one eye if let alone for a long time may produce amblyopia from

disuse, and the operation when ultimately performed may not be followed by a good visual result.

Traumatic cataract may be followed by the formation of a secondary capsular cataract, or after-cataract, which must be dealt with by capsulotomy.

Opacities of the Lens. All opacities of the lens and its capsule are included under the name cataract. This name is a relic of the time when the nature of these opacities was not understood, and no distinction could be made between the different kinds of opacities. Usage has established the name so firmly in our nosology that it will probably be permanently retained, and great confusion would follow any attempt to abandon it.

The following classification includes the forms of cataract met with clinically:

Capsular opacities.	Anterior capsular.	Congenital. Acquired.
	Posterior capsular.	
	Secondary, or after-cataract.	
Lenticular opacities.	Stationary (partial).	Zonular cataract. Circumscribed opacities.
	Progressive.	Nuclear. Cortical.

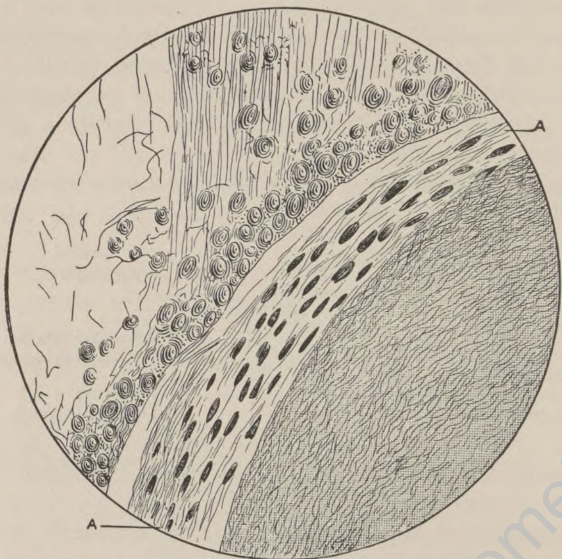
Capsular cataracts are, as the name implies, opacities confined to the capsule of the lens.

Anterior capsular or polar cataracts are of two kinds, conveniently considered as congenital and acquired. We have seen that the anterior capsule has an epithelial lining on its lenticular surface. Anterior capsular cataract consists in a proliferation of these epithelial cells, which become elongated and form a mass of opaque tissue resembling fibrous tissue, lying between the clear capsule and the clear lens, neither of which is affected by it. (Fig. 268.) This mass is not fibrous tissue, for it develops from epithelial (epiblastic) cells. In the congenital form of anterior capsular cataract the condition is due to some developmental error not yet determined. In the acquired form, which arises, as a rule, during childhood, the first step is an ulceration of the cornea, which perforates and allows the aqueous humor to escape and the lens to fall forward and apply itself to the opening. By contiguity inflammation is set up in the capsular epithelial cells, and they proliferate. Closure of the corneal opening and reaccumulation of the aqueous humor results in replacement of the lens to its natural position. We can see the corneal opacity, suggesting the cause, and sometimes a thread of tissue may be seen connecting the corneal opacity with the lenticular one. In these cases there is, in addition to the epithelial mass beneath the capsule, usually a plug of connective tissue deposited on the anterior surface of the capsule (pyramidal cataract).

Posterior capsular or polar opacities have a very different origin and are always congenital. In fœtal life a bloodvessel, the hyaloid

artery, runs through the vitreous, connecting the optic disk and the posterior surface of the lens. This usually atrophies, but sometimes a remnant of tissue remains at the site of the juncture of the hyaloid artery and the posterior surface of the lens, and this constitutes

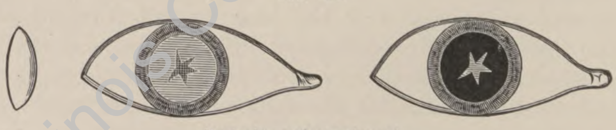
FIG. 268.



Epithelial proliferation (at A A) beneath the capsule. From a case of choroidal sarcoma. To show the histology of anterior capsular cataract. × 100. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

posterior capsular cataract. (Fig. 269.) As will be seen, it differs from anterior capsular cataract by lying on the surface of the capsule farthest from the lens, and in being fibrous (mesoblastic) instead of epithelial. Sometimes the hyaloid artery does not disappear, but remains in its entirety. Capsular cataracts are stationary.

FIG. 269.



Posterior polar cataract.

Lenticular opacities are far more common than capsular opacities, so much so that the word cataract, unless qualified, is usually taken to mean an opacity of the lens itself.

Lenticular opacities are caused by anything that interferes with the normal growth of the lens. In this way errors of development, such as faulty, delayed, or imperfect development, senility, constitu-

tional diseases, and other diseases of the eye, especially the diseases of the uveal tract, which is especially concerned in the nutrition of the eye, act as causes of cataract. The low-grade choroidal inflammation, described in the living as "choroidal disturbance," brought about by eyestrain attending uncorrected or improperly corrected errors of refraction, is held to be a potent cause of lenticular cataract. The writer has stated the causes in the foregoing manner because that idea best explains the greater relative frequency of cataract in the illiterate and ignorant classes—*e. g.*, the negro race—in whom errors of refraction are relatively rare and use of the eyes in a manner calculated to cause eyestrain very unusual. Cataract is common in glassblowers and others whose occupation exposes them to high temperatures, probably because these excessive temperatures cause

FIG. 270.

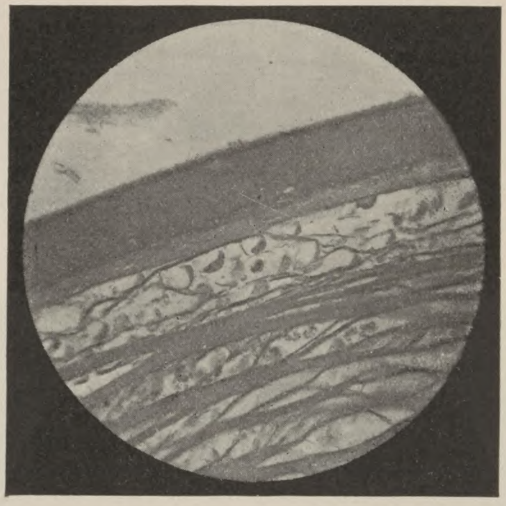
Beginning cataract. The nucleus is beginning to shrink, and a number of separations in the fibre are shown. These separations are filled with granular matter which stains deeply. × 100. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

similar conditions of the choroid. The manner in which these causes act is understood best when we consider the

Pathology of Lenticular Opacities. The lens grows, as we have seen, by the formation of new fibres from the layer of epithelial cells which underlie the anterior capsule. These cells become fibres by a process of elongation, and go to make up the cortical portion of the lens. The tendency of the capsular epithelium to take on this fibrous structure has been seen in considering anterior capsular cataract. The nucleus is the oldest part, and by contraction and loss of fluid gradually becomes more dense and smaller. If the growth of the

lens is arrested by senility or by any other cause which interferes with its growth, the nucleus continues to shrink, and small spaces are created by its drawing away from the cortex. (Fig. 270.) These

FIG. 271.



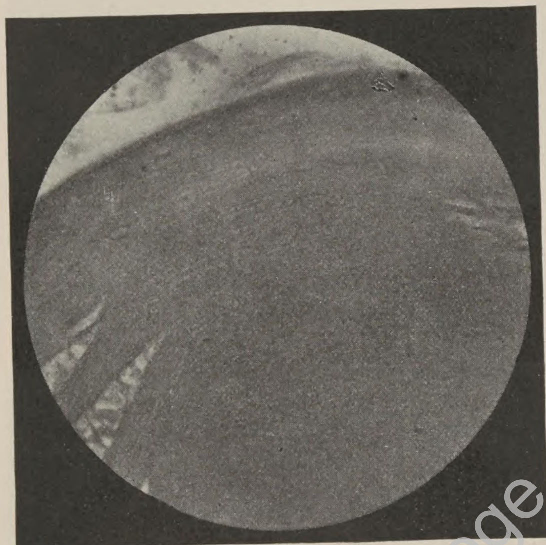
Senile cataract. The cortex is homogeneous. Deeper in, the fibres are separating through shrinking of the nucleus, and the spheres of Morgagni are forming. × 100. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

FIG. 272.

Senile cataract. Separation of fibres and formation of spheres of Morgagni. × 200. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

spaces, therefore, as a rule, lie in the perinuclear region, especially toward the equator. These spaces become filled with an albuminous fluid, like serum, which, while clear, appears opaque as compared to the fibres, on account of the difference in the index of refraction of the two. Thus the first appearance of cataract when seen in its period of development is of opaque lines running from the circumference. By oblique illumination these lines appear gray, but by reflected light they appear black, as they interfere with the light reflected back from the fundus. The albuminous fluid in the spaces between the fibres coagulates to form drops, called the spheres of Morgagni. (Figs. 271 and 272.) The lens fibres next to these spaces

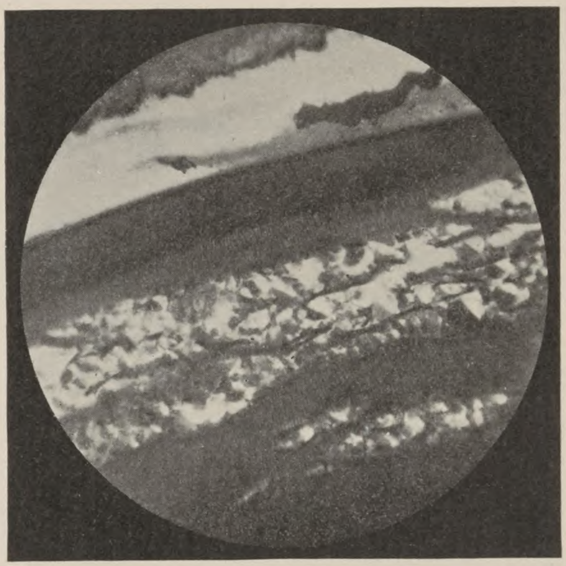
FIG. 273.



Senile cataract. The fibres are beginning to break up, and the lens looks granular and homogeneous. × 100. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

become clouded by minute fatty drops, and swell and become more and more opaque by fatty degeneration and imbibition of the fluid. (Figs. 273 and 274.) This process may be arrested by the removal of the cause, and, except in senile cataract, the lens resumes its normal growth. The nucleus, being more dense, is usually the least affected by the process, and in many cataracts it is yellow and comparatively clear, while the cortical portion is milky and opaque. If the cataract progresses to complete opacity of the lens, the same fluid is secreted between the lens and the capsule, and tends to separate them. (Figs. 275 and 276.) This makes removal of the lens easier, and explains why we prefer to delay the extraction of progressive cataracts until they are fully opaque or "ripe." When

FIG. 274.



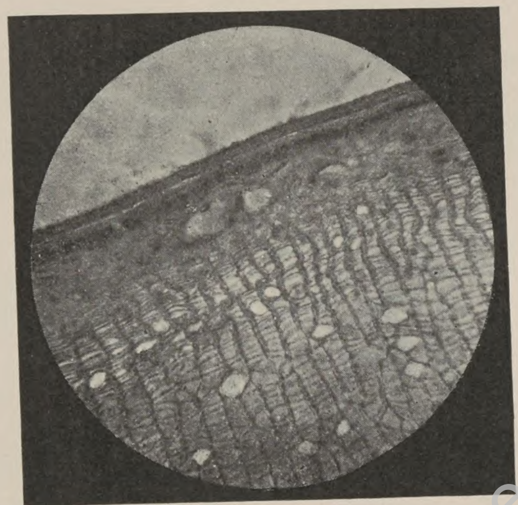
Senile cataract. Showing separation of fibres and granular débris. Fibres cut transversely. × 100. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

FIG. 275.

Cataract following iridocyclitis. Showing softening of the cortex and separation of the outer layers of the nucleus. × 15. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

this stage is reached, the whole lens contracts until the increase in size, due to swelling of the fibres of the cortical portion, is lost, and the lens regains its original size. The degeneration of the fibres continues until the cortical portion is converted into a pultaceous opaque mass, which becomes finally perfectly fluid, and in this fluid the still firm nucleus floats. This condition is known as hypermature or Morgagnian cataract. The fluid portion may become clear and the lens in a measure regain its transparency, but not its consistency, and some improvement in vision takes place. The nucleus never entirely disappears, although it becomes progressively smaller. Cholesterin crystals may form in the fluid part of a hypermature cataract.

FIG. 276.



Senile cataract. The cortical layers beneath the capsular epithelium are softened, and several large swollen nucleated cells show. Below these are a few vacuoles between the fibres. × 200. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

The symptoms of cataract consist principally in diminution of the acuity of vision. In the early stages the presence of areas in the lens differing in refractive index from the lens itself may give rise to polyopia, or multiple vision, but this is not very common. The eye surgeon is usually consulted on account of dimness of vision. If the opacity is peripheral, it causes very little inconvenience as long as the central portion of the lens remains clear. Such a person sees best with a contracted pupil when the opaque portions are hidden behind the iris. On the other hand, if the opacity is central, the patient sees best when the pupil is dilated—*e. g.*, at night—when the retraction of the iris permits the passage of rays of light through the clear peripheral portions of the lens. As the opacity progresses the vision is poor under all circumstances, and the pupil assumes a noticeably gray or white color. The vision is never entirely lost from

uncomplicated cataract; that is, the patient can always not only perceive light, but can tell the direction whence it comes. If a patient with cataract is unable to determine the location of a lighted candle at a distance of fifteen feet in a moderately darkened room, we may be sure that some lesion of the retina or optic nerve is present, and that removal of the cataract will not be attended with anything like a perfect restoration of vision.

The impairment of vision which is observed in the development of a cataract is sometimes due to another cause than opacity of the lens. It frequently happens that prior to the development of any considerable opacity the increasing density of the lens renders its refractive index higher and its action as a lens stronger. The result of this is to make the eye myopic, and this is the condition which constitutes so-called "second sight." A previously emmetropic or hyperopic eye becomes myopic, and if the patient is at an age when presbyopia has appeared—and this is usually the case, since this condition is seen oftenest preceding the development of senile cataract—he finds that he is enabled to dispense with his presbyopic glasses and read with the unaided eye. The distant vision is, however, reduced. Careful examination of such an eye will seldom fail to show commencing cataractous degeneration of the lens. An attempt to correct this newly acquired myopia by glasses is not very satisfactory, for although the patient may read the letters on a test-card with much greater fluency, for some reason the glasses do not seem to render him much practical aid in vision. The writer has frequently seen cases of this character in whom glasses would raise the distant vision from 20/100 to 20/50, or more, but who preferred to be without them. For their influence in relieving eyestrain and promoting nutrition of the eye, these glasses should be carefully fitted and worn.

The **course** of cataract is variable. We may except the different forms of stationary cataract, in which there is no tendency to change, and speak only of progressive cataract. The tendency is for the opacity to advance, but this occurs at a very variable and uncertain rate. We are, therefore, not able to prognosticate with any certainty the length of time which a given case will take to arrive at maturity and be ready for operation. The process may advance rapidly for a while, and then remain stationary for years, or, after a long period in which no progress is made, suddenly advance rapidly. In case both eyes are affected, we can argue with some degree of confidence that the last eye to be affected will run the same course as the first one did, but there are many exceptions to this. A plain statement of all these facts had better be made to the patient or the patient's friends at first. In a general way, it may be said that about two years is an average time for a senile cataract to arrive at maturity, while the progressive forms of cataract in younger people are apt to advance more rapidly and on the whole with greater uniformity. The value of this statement lies in the fact that we may inform the patient that it is not likely that the eye will be ready for operation

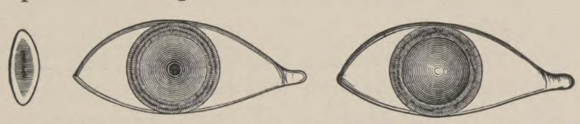
in less than two years, nor is it apt to be longer than three or four. But this statement should be made only as a mere approximation, since there is no way of arriving at a more accurate prognosis.

Progressive cataracts follow a course in their development which admits of division into four well-defined clinical stages:

1. Incipient cataract. In this stage second sight may be present, but the opacity is recognizable, especially with the ophthalmoscope. No other changes have yet occurred, and vision is present to a useful degree.
2. Intumescent or swollen cataract. The lens is now more opaque, although clear areas may still be found; but the fibres are swollen, the lens is larger than normal, the iris is pushed forward, and the anterior chamber is shallow.
3. Mature cataract. The lens has resumed its normal size and is opaque throughout.
4. Hypermature cataract. The cortical portion has undergone softening, or even liquefaction, and may have cleared up to some extent.

Diagnosis. The diagnosis of cataract is to be made by careful examination with both oblique illumination and with the ophthalmo-

FIG. 277.



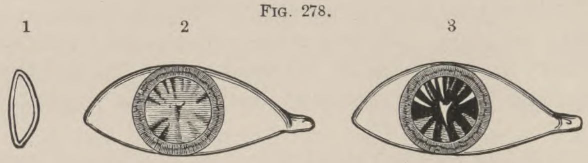
Nuclear cataract. 1. Section of lens; opacity densest at centre. 2. Opacity as seen by transmitted light (ophthalmoscopic mirror), with dilated pupil. 3. Opacity as seen by reflected light (focal illumination). The pupil is supposed to be dilated with atropine.

scope, and should aim to determine not only the presence of lenticular opacity, but also the precise location, stage of development, and kind of cataract, together with the cause, if possible, and the condition of all of the other ocular structures. It is important to examine the lens with the pupil fully dilated by means of a mydriatic. It is extremely important, in cases seen early, to make a careful examination of the fundus of the eye, since the opportunity to do this may soon be lost, and a knowledge of the conditions within the ball is highly advantageous in arriving at an intelligent opinion as to the cause and course of the cataract, as well as the chances for a good visual result following operation.

By oblique illumination opacities in the lens appear as gray dots, masses, or streaks against a black background. (Figs. 277 and 278.) If small and situated deep in the lens or very near the equator, they may not be visible at all by this method, and their density is, as a rule, not easy to determine. Opacities in the anterior layers of the cortex or on the anterior capsule are easily seen in this way, and we can also judge of the depth of the anterior chamber and the condition of the iris as to color, motility of the pupil, etc.

We determine when a cataract is mature by concentrating light on it from the side (oblique illumination). If the cortex is not opaque, the iris throws a very marked shadow through the clear outer

FIG. 278.

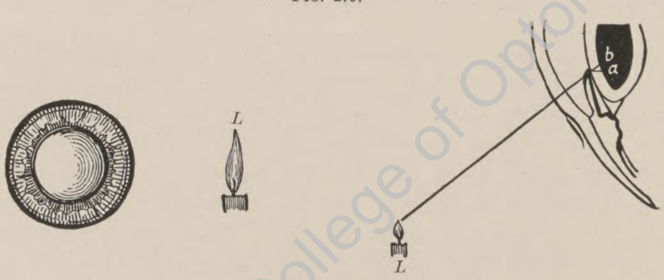


Cortical cataract. References as in preceding figure.

layers on the opaque central portion of the lens. (Fig. 279.) If the lens is fully opaque (mature cataract), no shadow is thrown.

By ophthalmoscopic examination opacities appear black against a red background, and on the whole are more easily seen. We can judge very well of their density by this method according to the degree of blackness. Faint nuclear opacities can be detected which escape being seen by oblique illumination altogether. Except in patients whose eyes show glaucomatous tendencies, the pupil should always be dilated with a weak and evanescent mydriatic, such as a 2 per cent. or 4 per cent. cocaine solution, or a 1 per cent. euphthalmine solution, or a combination of the two in a 5 per cent. or 1 per cent. solution. Otherwise peripherally located striæ, the form in which senile cataract often commences, will be hidden from view by the iris. (Fig. 278.) The dilatation of the pupil also enables us to judge

FIG. 279.



L. Shadow of the iris seen from in front in immature cataract. *L* Equals source of light. *L L.* Shadow of the iris upon the lens seen in schematic cross-section. *L.* Equals source of light. (FUCHS.)

of the condition of the pupil as to its motility and the presence of posterior synechiæ. The extent of central opacities may be clearly defined through the dilated pupil, and the condition of the fundus studied through the clear peripheral portions of the lens.

The location of the opacity, if small, can be made out by ophthalmoscopic examination by observing the motion of the opacity in regard to the motion of the pupillary edge of the iris. If the opacity is on the anterior capsule of the lens or in the anterior portion of the

cortex, it will appear to move in the same direction as the eye moves, *i. e.*, upward if the patient looks upward. This is determined by

FIG. 280.

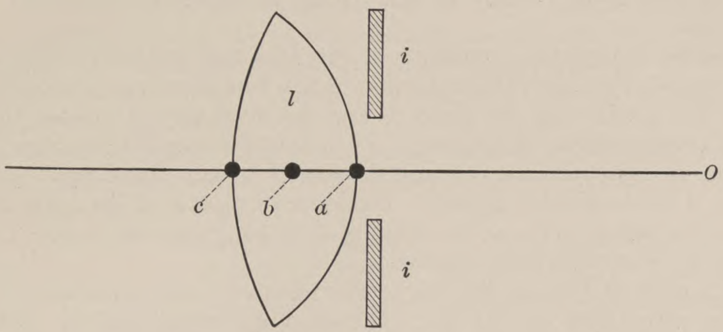


FIG. 281.

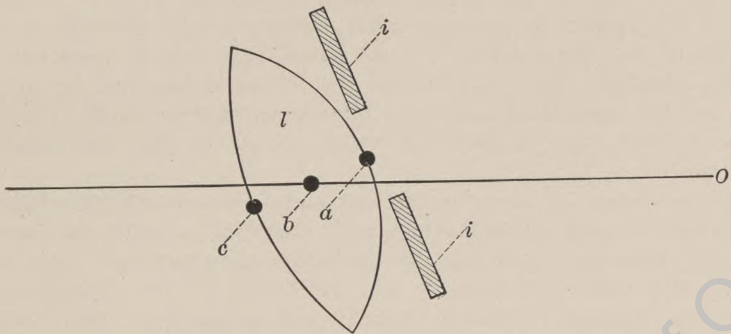
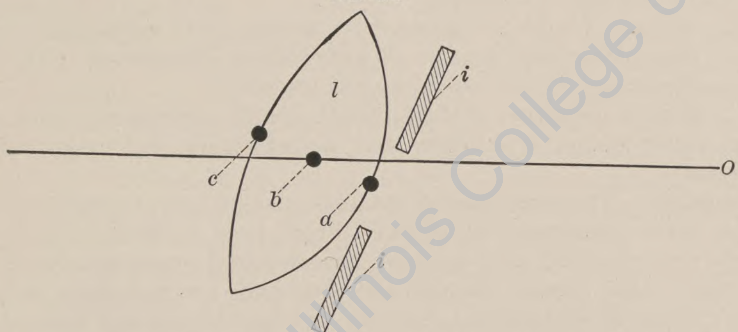


FIG. 282.



Diagrammatic representation of method of detecting location of lenticular opacities by their movement in relation to the movement of the edge of the pupil. Fig. 280, the eye is looking straight ahead. Fig. 281, the eye is looking upward. Fig. 282, the eye is looking downward. *i.* Iris. *l.* Lens. *O.* Observer. *a.* Anterior opacity. *b.* Central opacity. *c.* Posterior opacity. *Oc.* Line of vision of observer.

noting its distance from the edge of the pupil. If the opacity lies near the posterior surface of the lens, it will appear to move in the

opposite direction to the movement of the eye—*i. e.*, if the patient looks upward, the opacity appears to move downward. A central opacity—that is, one equally distant from the two surfaces—will move very little or not at all. This is shown in Figs. 280, 281, and 282.

Besides noting the condition of the lens and anterior segment of the eye, we should, if the condition of the lens permits, examine carefully for opacities in the vitreous and for evidences of disease in the optic nerve, retina, and choroid. The latter being the nutritive coat of the eye, the presence of any disease of it will throw light on the cause of the lenticular disease. Evidence of disease of the optic nerve or retina will govern us in arriving at a prognosis as to the visual result to be attained by operation.

If a view of the fundus cannot be obtained, the condition of the deeper structures should be determined by measuring the field of vision. This can be done even in mature cataracts by means of two lighted candles. It has been stated that cataract alone never causes loss of the ability to perceive light; hence, in the absence of other disease of the eye a fairly normal visual field can be demonstrated by the candles. Not only should the limits of the field be mapped out, but the central region studied in the same way for the existence of scotomata, the presence of which would modify the prognosis materially.

The condition of the conjunctiva should be noted for evidences of present or past inflammation, and, above all, should we carefully examine into the condition of the lacrymal apparatus, both at its ocular and nasal extremities, and determine if the apparatus performs its drainage functions properly and is free from inflammation. The lacrymal apparatus is probably the most fruitful source of post-operative infection of the eye.

The eyeball should be palpated to determine if tenderness exists and if the intra-ocular tension is increased or diminished. The significance of these conditions is stated elsewhere.

The general condition of the patient's health, secretions, urine, etc., must all be looked into before our investigation and diagnosis are completed.

Prognosis. The prognosis of cataract, except from operative treatment, is for permanent blindness. From time to time other treatments are exploited, but none so far advanced possesses any value. Massage of the eyeball through the closed lids has had some support from reputable professional sources, but has practically been abandoned as without value. Other treatments by the instillation of certain drugs, such as cineraria maritima and other substances, and various "absorption treatments," have emanated from unprofessional sources or from charlatans, for commercial reasons, and are also worthless.

Spontaneous clearing of cataractous lenses has been occasionally reported by observers of repute, but is extremely rare. Risley has

called attention to the fact that some, at least, of these cases were not lenticular opacities, but inflammatory deposits between the posterior capsule and the vitreous, and were of choroidal origin. It is easy to understand the disappearance of these products, but in a few instances clearing up of true lenticular opacities has been seen to occur. These cases are ophthalmological curiosities.

Cataract is sometimes "cured" by the spontaneous or traumatic dislocation of the lens.

In cataracts which are allowed to advance to hypermaturity, the fluid cortical substance sometimes becomes sufficiently clear to permit some restoration of vision.

The prognosis from operation is good. In uncomplicated cataract the operation of removal of the lens should yield a good visual result in about 90 per cent. of cases. The prognosis is rendered less favorable by complicating disease of the eye and by certain depressed states of nutrition, as in diabetes. A careful examination, as was mentioned under the head of Diagnosis, will lead to the detection of these conditions, and the prognosis can be modified accordingly.

In considering the question of operation for cataract on patients who, for some incurable local or general disease, such as suppurative condition about the eye, trachoma, or diabetes, nephritis, pulmonary phthisis, etc., offer a bad prognosis, the matter should be explained fully to them in regard to the risk that they run of losing the eye, and they should then be advised to have the operation performed, for the reason that in the event of failure their condition is no worse than if they were not operated on, that is to say, they are blind in either event.

The clinical varieties of cataract have been stated in the preceding pages.

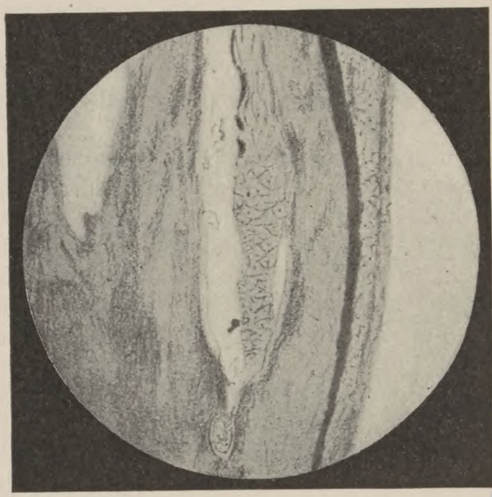
Capsular Opacities. 1. Anterior capsular or polar cataract is either congenital or is acquired in infancy as the result of corneal ulceration and perforation. There is a subcapsular hyperplasia of the epithelium, and in the acquired form frequently a deposit of lymph on the anterior surface of the capsule, forming what is sometimes called pyramidal cataract. From this a filament of organized lymph may sometimes be seen to run to a small corneal opacity.

2. Posterior capsular or polar opacities lie on the posterior layer of the capsule, and are caused by failure of absorption of the tissue where the fœtal hyaloid artery joins the lens. Sometimes the hyaloid artery persists, and may be filled with blood or may have its lumen obliterated and appear as a connective-tissue filament running from the optic disk to the lens.

3. Secondary or after-cataract is the name applied to portions of the capsule left behind in the pupillary space after removal of the lens. Unless the lens is removed in its capsule, a secondary cataract always remains. This may be so thin as not to interfere with vision, and then is of little clinical importance. On the other hand, it may be quite dense and augmented by particles of lens matter enclosed

between the posterior capsule and the remains of the anterior capsule, and further thickened by a deposit of lymph thrown out from the iris during the iritis which often follows operation for cataract. (Fig. 283.) If the central portion of the secondary cataract is suf-

FIG. 283.



Remains of lens after a normal cataract extraction. Showing swollen lens fibres surrounded by iris and ciliary exudate. From an eye which was enucleated on the tenth day for iridocyclitis. × 15. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

ficiently opaque to interfere with vision, it requires treatment by the operation of capsulotomy.

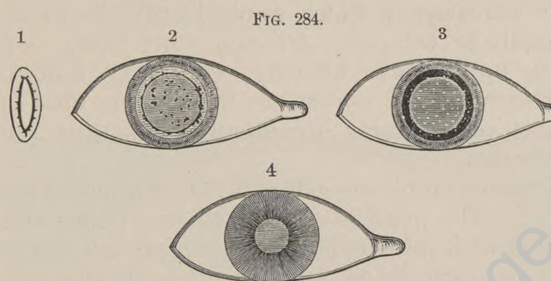
Lenticular Opacities. 1. Stationary opacities of the lens are of various kinds:

(*a*) Circumscribed opacities of the lens may follow a penetrating wound confined to a small area. We cannot count on cataracts of this character remaining stationary, but the clinical fact is that they sometimes do, and may even disappear. It has been said that in some of these cases the wound in the capsule is minute and instantly closes, keeping out the aqueous humor. The opacity is then due to mechanical disturbance of the lens fibres. Circumscribed opacities of other than traumatic origin are sometimes seen, and an adequate explanation of them is hard to give. The most common form is that in which opaque masses and spiculæ exist in the lower and inner quadrant of the lens, as described by J. L. Thompson. This is the form of senile cataract which is most apt to remain partial for a long time. Other forms are punctate opacities, which are multiple and may occupy almost any position; stellar opacities, which are unusual in the posterior central region and often associated with retinitis pigmentosa or choroidal disease, and vitreous opacities; central cataract, an opacity lying near the centre of the lens; and

spindle or axial cataract, which consists of a fusiform opacity running from before backward through the centre of the lens, with the thickest portion at the centre.

(*b*) Zonular cataract is a common form of congenital or infantile cataract. From arrest of development or growth of the lens a cataract begins to form in the perinuclear region. The cause of this arrest of development or growth being removed, the opacity ceases to progress, the surrounding and subsequently formed lens substance being clear. The condition is then that of an opaque shell lying in the lens, enclosing a clear nucleus and enclosed by a layer of clear cortex. A repetition of the process at a later date may result in the formation of a second opaque zone, separated from the first by clear lens tissue. Zonular cataract may become progressive by the overlying layers becoming opaque. This may be foretold when peripheral opaque spiculæ are seen lying in front of the opaque zone. These spiculæ from their relation to the opaque zone are called "riders." By oblique illumination this form of cataract resembles an immature progressive cataract. The ophthalmoscope shows the centre to be slightly clearer than the peripheral portions of the opaque area, and surrounding the opacity a clear area through which a bright reflex is obtained and the fundus can be seen. (Fig. 284.) The appear-

FIG. 284.



Lamellar cataract. 1, 2, 3. As before. 4. Shows slight grayness of the undilated pupil, owing to the layers of opacity being deeply seated.

ance of clearness in the centre is due to the fact that the opaque layers are further separated here, while at the edge of the opacity the two opaque layers are so close that they have the effect of one thick layer. Zonular cataract is associated so often with a history of convulsions due to rickets that they are generally believed to bear a causal relation to it.

(*c*) Anterior and posterior cortical opacities are sufficiently described by their name. They are lenticular opacities, in contradistinction to anterior and posterior capsular cataracts, and their special cause is not understood.

2. Progressive cataracts include the vast majority of opacities of the lens. While sometimes seen in young adults, they usually affect persons over forty years of age, and constitute what are spoken of

as senile cataracts. It must not be forgotten that progressive cataracts may be seen in infancy—indeed, at any age. Before the age of thirty years cataracts partake of the characteristics of the normal lens in that they are composed largely of the outer soft cortical portions, and of relatively little nucleus. Senile cataracts, on the other hand, have relatively larger nuclei, and are called hard cataracts, in distinction from juvenile or soft ones. There is always, even in the aged, a layer of softer cortex around the nucleus, although this is less, as a general rule, the older the patient. Clinically, we distinguish soft from hard cataracts, when mature, by the fact that the former are of a more uniform and greater whiteness, while the latter may be quite brown, due to the larger brown nucleus. A very large and dark nucleus causes what is called black cataract, and such cataract may be all nucleus and never become mature. Senile cataracts possess four fairly well-defined stages, as has been mentioned: 1. Incipient. 2. Intumescent. 3. Mature. 4. Hypermature.

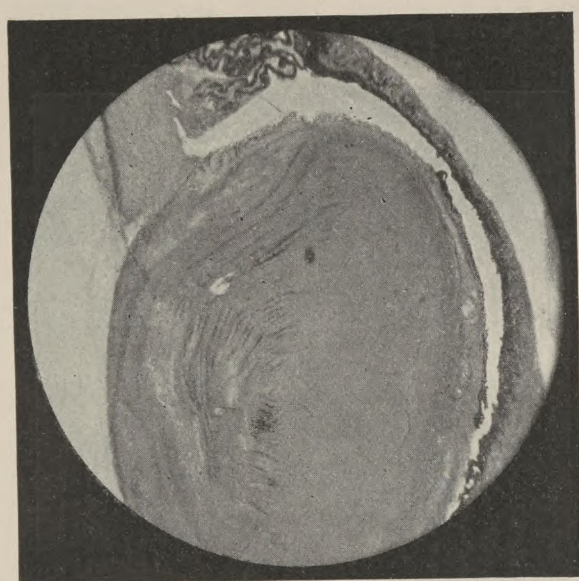
Complicated cataract is the term applied to cataracts accompanied by other intra-ocular disease of such a nature as to give rise to complications during the performance of the operation for their removal. Thus corneal opacities which obscure the view of the field of operation, iritic adhesions which bind the lens in place, fluidity of the vitreous, rendering its escape likely during the operation, are conditions the existence of which would justify the name of complicated or complicate cataract. On the other hand, conjunctivitis, dacryocystitis, optic nerve atrophy, etc., may militate seriously against the success of the operation, but custom does not sanction the designation of cataracts accompanied by these conditions as complicate cataract.

A special form of complicate cataract is that which is seen following neglected iritis. The pupil is contracted to a point, and this small opening is filled with an exudate so intimately adherent to the capsule, which is also opaque, as to constitute a capsular cataract. The iris is frequently bound to the capsule by this membrane over its whole extent, and the lens is often opaque. Iritis of this severity is frequently accompanied by cyclitis, as a result of which the intra-ocular tension is lowered.

Etiology of Cataract. To reserve until now a consideration of the causes of cataract is merely to recite as causes conditions whose *modus operandi* has been discussed and will readily be understood.

Congenital cataracts are due to defective development, which in turn is due to some local or general disease of the mother. Syphilis, tuberculosis, or other diathetic condition may act in this way. The defective development of one or more of the products of twin or triple pregnancy may express itself in this form, although seldom unassociated with other physical and possibly mental defects. Trauma, either directly or indirectly to the lens, is a cause. General diseases which affect the nutrition, and senility through a similar cessation of growth, have been discussed as causes. Local diseases of the eye,

FIG. 285.



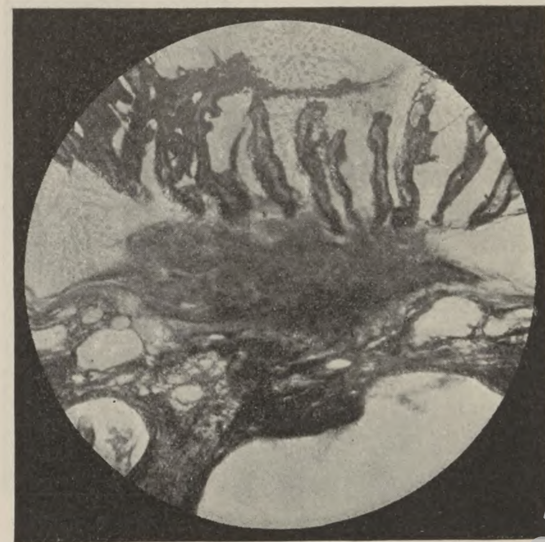
Shrinking lens in a case of iridocyclitis. × 15. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

FIG. 286.

Remains of the lens in phthisis bulbi following iridocyclitis. × 15. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

particularly choroidal disease, are fruitful causes. (Fig. 285.) Choroidal cataract is a well-defined variety, and is seen often in young

FIG. 287.



Section from a case of phthisis bulbi with ossified choroid, showing the lens converted into a mass of fibrous tissue. × 15. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

FIG. 288.

Wrinkling of lens capsule and softening of cortex under a large iris adhesion (artificially separated). From a case of iridochoroiditis. × 100. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

persons. In this form the lens is very apt to undergo calcareous infiltration, and presents a porcelain-like whiteness. Diseases of the eye which terminate in atrophy of the globe nearly always produce cataractous lenses. (Figs. 286 and 287.) In the same way congenitally microphthalmic eyes have opaque lenses.

Eyestrain from uncorrected errors of refraction acts as a cause by producing a choroidal disease of low type but long duration, and thus interfering with the nutrition of the lens. Iritis sometimes causes cataract by the traction of the resulting synechiæ. We cannot say whether this acts by first producing a rent in the capsule, or by the disturbance due to traction alone, acting just as a contusion or massage of the lens does. (Fig. 288.) In chronic glaucoma the lens usually becomes opaque in time, due to nutritive disturbances in the eye which result from the glaucomatous process. (Fig. 289.)

FIG. 289.

Deformity of lens in a cataract from chronic glaucoma. × 15. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

Treatment. The treatment of cataract varies with the variety. It will be convenient, in order to avoid repetition, to describe in a separate section the operations to be performed for cataract.

Capsular Opacities. Anterior or posterior capsular and polar cataracts are, as a rule, so small that they do not seriously interfere with vision, and may be let alone. In case they do, and treatment is necessary, we have no means of attacking the capsular lesion alone, but discission of the lens must be performed and the case subsequently managed like a soft or traumatic cataract. Iridectomy,

often of value in some forms of partial cataracts, is usually valueless in anterior and posterior polar cataract.

Secondary or after-cataract is to be treated by operation if it interferes with vision to any considerable extent. The treatment is by the operation of capsulotomy.

Lenticular Opacities. Stationary (partial) cataracts require different treatments, according to the impairment of vision which they cause and the condition of the other eye.

Zonular cataract is, as has been said, the most common variety of lenticular cataract seen in children. If the opaque portion is small in diameter, it will be found that dilatation of the pupil will so expose the clear peripheral portion of the lens as to permit very satisfactory vision through it. In such a case, while permanent dilatation of the pupil by continuous instillation of a mydriatic may be practised, it is inconvenient and not free from the possibility of a deleterious effect on the eye or general health of the patient. For these reasons it is preferable to make a portion of the clear periphery of the lens available permanently for vision by the performance of an iridectomy. This should be done in an upward direction, and the coloboma made of moderate size, but extending to the base of the iris. The refraction should then be carefully corrected and lenses given for constant use. If both eyes are affected, both should be operated upon. If only one eye is affected and the vision of the other eye is good, this operation should not be performed. The affected lens should be treated by solution or not at all. If the cataract shows a tendency to progress, the operation for solution (discission) should be performed, as iridectomy will afford only temporary relief. In the absence of any contraindication, the treatment by solution should be performed in all cases as affording the best visual result. We can count on securing normal or nearly normal vision, and can be sure that the effect is permanent. Both eyes should not be operated on at once, as some accident or intercurrent disease may cause the loss of both. By operating on the two eyes at different times, we profit in the second eye by the experience gained in treating the first, as to the peculiarities of the patient and the behavior of the eye as to reaction, etc. The same rule applies to all double cataracts.

Circumscribed stationary lenticular opacities should be treated on the same principles. If the fellow eye is good, no operation is necessary on the affected one. If it is defective or has been removed, the question of operation will be determined by the amount of visual impairment, the presence of complications, and the help afforded by correcting lenses. The operation will be that of solution or extraction, according to the patient's age and the consequent hardness of the lens and the size of its nucleus.

Progressive lenticular opacities should, if slight, be treated by what means we have to retard the progress. Careful correction of refraction, including the acquired myopia, and measures to improve

the general health, together with the treatment of any pronounced dyscrasia, such as diabetes, nephritis, etc., will often render excellent aid in retarding the development of a cataract. Correction of the refraction lessens the choroidal disturbance caused by eyestrain, and in this way contributes to better nutrition of the lens. The improvement of vision which lenses bring about is also a source of the greatest satisfaction in some cases. There is plenty of evidence to show that these measures are of undoubted value, and they should be tried faithfully. We have spoken of the futility of "absorption treatments" and drugs. Alteratives undoubtedly exercise a beneficial influence in some cases.

As in zonular cataract, so in progressive lenticular cataracts, dilatation of the pupil by mydriatics or the performance of an iridectomy may assist in obtaining temporary improvement of vision. As a rule, these measures are worthy of trial only when the vision of the other eye is seriously impaired or altogether absent. In behalf of iridectomy, it may be said that its performance at some time before extraction of the lens renders the latter operation easier and safer.

In some cases contraction of the pupil by myotics (pilocarpine and eserine) will be found to improve the vision when the centre of the lens is clear or tolerably clear.

In deciding on the use of mydriatics or myotics, it is best to test the patient's vision with the eye under their influence at his occupation or in going around, to see if his condition is improved, before suggesting their use or advising an iridectomy, for, although they may help the patient to see more letters on the test-card, they may not render his vision any more useful in any other way, and their use would be wholly unsatisfactory.

The utility of ripening operations for hastening the maturity of lenticular opacities is questionable. Most operators prefer to extract an unripe lens rather than resort to them.

The operation of removal of the lens in progressive opacities is, as a rule, best deferred until maturity. This is not necessary in soft cataracts—*i. e.*, in patients under thirty-five years of age, in whom the operation of solution is applicable. In senile cataracts it is best to wait until maturity. The lens is then removed by extraction. When the patient has a mature cataract in one eye and the lens of the other eye is clear, it is well to advise removal of the cataract for the following reasons: it improves his field of vision by enabling him to see at least large objects on the affected side, and this prevents him from being run into or from running into objects and other persons. The eye may, from accident to the other or the development of cataract or other disease in it, be at some time his main dependence, and the vision will be better if it is allowed to be exercised by removal of the lens than if it is excluded from the visual act for years maybe by the cataract. Amblyopia from disuse may develop in an eye which is affected with cataract, particularly in children.

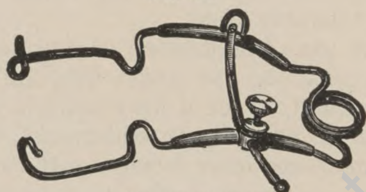
Complicated cataracts are a law unto themselves. In general, if the other eye possesses good vision, complicated cataracts had better be let alone. If the other eye does not possess and cannot be made to obtain useful vision, operation should be resorted to, even if the chance of success is small. Solution or extraction should be performed, according to the age of the patient.

The operative treatment of cataract comprises several operations. For hastening maturity in unripe cataract, various ripening operations have been devised. For the removal of opaque lenses, the operation may be that of depression, or solution, or extraction. For the membranous opacities classed as secondary or after-cataracts, the operation of capsulotomy may be done. For occlusion of the pupil after the removal of the lens, various operations on the iris, such as iridectomy, iridotomy, etc., may be required.

General Considerations. See Chapter XIV.

The operation of depression was formerly extensively practised, but was abandoned because the eyes were subsequently lost, either from glaucoma or iridochoroiditis, caused by the lens acting as an

Fig. 290.

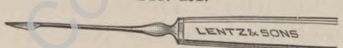


Eye speculum.

Fig. 291.

Fixation forceps.

Fig. 292.



Hayes' knife needle.

Fig. 293.

Broad paracentesis needle.

irritating substance. The operation consists in displacing the lens downward and backward into the vitreous. The instruments required are a wire speculum (Fig. 290), toothed fixation forceps (Fig 291), and a broad needle (Fig. 293). The lids being separated by the

speculum, the conjunctiva and subconjunctival tissue are grasped with the fixation forceps to steady the eye, and the needle entered either at the margin of the cornea or in the sclera behind the iris. It is placed against the posterior surface of or thrust into the lens, and

Fig. 294.

Straight cataract needle.

by a lever-like action forces it downward and backward. The needle is then carefully and quickly withdrawn. The immediate results of this operation are brilliant, unless the lens rises to its normal position again. The ultimate results are usually loss of the eye from subsequent inflammation or glaucoma. The operation is only justifiable in those so feeble from age or disease that they would probably not do well under the operation of extraction, but to whom it is desired to give some sight during their short remainder of life.

The operation of solution or discission is applicable to cataracts in young people. The age limit is variously stated as from fifteen to thirty-five years. We would prefer it on patients under thirty years of age. The operation consists in making an opening in the anterior capsule and lens, and submitting the latter to the action of the aqueous humor. We have seen that this causes the lens fibres to become opaque, swell, and ultimately absorbed. Anterior and posterior capsular cataracts (if they require treatment), zonular cataract, progressive juvenile cataracts, and other opacities of the lens in young people, are to be treated by this operation.

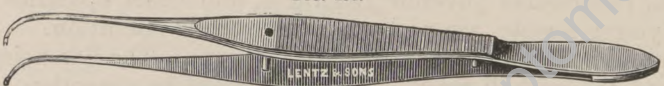
The pupil is first fully dilated with atropine. The lids being separated and the eyeball fixed, as in the preceding operation, a small needle, or a knife needle (Fig. 292), is thrust through the cornea well toward the periphery, and carried to the centre of the pupillary space. The point of the needle is entered through the centre of the capsule into the lens, and in withdrawing it the opening is slightly enlarged. The needle is quickly withdrawn from the cornea, without, as a rule, losing the aqueous humor. It is well at the first operation to make a very small opening, since we do not know how the lens or eye will react. Should no irritation show itself, and the change produced in the lens be slight, a freer opening may be made in a few days and in the same manner. A generous central T-shaped or crucial incision is a good form, and is followed usually by rapid swelling of the lens, which protrudes through the capsular opening and fills the anterior chamber with broken, swollen, and opaque fibres. These may be removed from the eye by simple linear extraction (*q. v.*). Pain in the eye and increased tension (glaucoma) necessitate this operation at once, and it promptly relieves these symptoms. If this is not done, the lens slowly absorbs. This process may be hastened by breaking down the larger masses by needling or

further opening of the capsule. Complete absorption requires from two to six months, and during this time the eye should be kept constantly under the influence of atropine. A drop of a solution of two to four grains to the ounce is instilled into the eye from one to four times a day. On account of the time it takes the lens to absorb, it is advisable to remove some of it by simple linear extraction when possible. The absorption of the lens usually leaves an after- or secondary cataract, which requires operation. After this the eye requires a high strong convex lens to enable it to see. When the lens has been partially absorbed as the result of discission, a method to hasten the attainment of vision has been proposed by Dr. G. C. Savage, and is as follows: A needle is introduced into the eye and the centre of the lens cleared by pushing the fragments toward the periphery. In this way a clear pupil may be obtained some weeks before it would be available by the process of absorption.

Operations of discission, and capsular operations may be considered "office operations," and can be done on a couch or with the patient sitting in a chair and resting his head against the body of the surgeon, who stands behind him. Subsequent rest is desirable, but subsequent immobility is not essential.

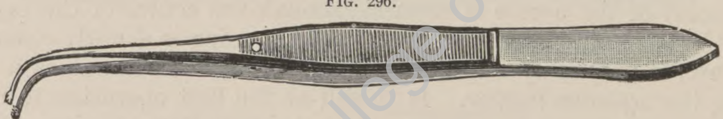
Ripening Operations. These consist in procedures whose essential part is the application of massage to the lens for the purpose of so disturbing the superficial portions that opacification of the outer cortical portion is hastened. Their main influence is exerted on the peripheral layers of the cortex anteriorly.

FIG. 295.



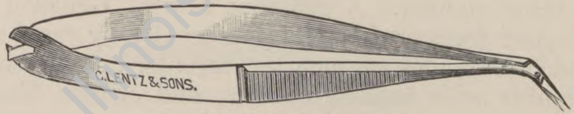
Iris forceps.

FIG. 296.



Capsule forceps.

FIG. 297.

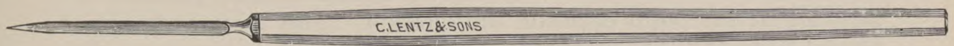


McClure's iris scissors.

Instruments. Speculum (Fig. 290), fixation forceps (Fig. 291), keratome or paracentesis needle (Fig. 293), blunt probe or trowel-shaped spatula, iris forceps (Fig. 295), and iris scissors (Fig. 297).

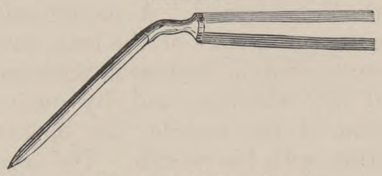
An iridectomy or simple paracentesis corneæ is done, and massage applied to the lens, either directly by the probe or spatula introduced into the anterior chamber, or the lens is massaged through the cornea. The movements are rotary and very gentle. Discission

FIG. 298.



Graefe knife.

FIG. 299.



Baeder's knife (right and left).

is also sometimes used for the purpose of ripening cataracts. The present view of the method may be well expressed in Knapp's words: "All these procedures have the disadvantage of being, in a number of cases, either totally or partially inefficient, besides adding to the removal of the cataract another surgical procedure which has not always proved harmless."

Most operators prefer to extract immature cataracts rather than resort to any method of artificial ripening.

Extraction of cataract is the term applied to operations whereby the lens or the greater portion of it is removed at one sitting. The different procedures may be grouped under the heads of (1) simple linear extraction, and (2) extraction of hard cataract.

Simple linear extraction is applicable to soft cataracts—*i. e.*, in persons under the age of thirty years—and to traumatic cataracts. It is performed as follows:

The pupil is dilated with atropine, local anæsthesia induced, the lids separated by a speculum, and the eye grasped with fixation forceps, as already described. A narrow keratome or broad needle is entered through the cornea just within its clear margin, and usually at the point nearest the external canthus. By pushing the instrument forward, and, if necessary, by a lateral movement in withdrawing it, an incision about 5 mm. long is made. The instrument should be withdrawn very slowly to permit gradual emptying of the anterior chamber, as rapid evacuation of the aqueous, especially if the tension of the eyeball is increased, may be accompanied by sensations which cause the patient to become alarmed and move. If the operation is undertaken as the primary operation on a soft cataract, the capsule may be incised by the same instrument which makes the corneal incision, by directing its point backward, or this may be done with a cystotome (Fig. 300) after withdrawing the

first instrument. Frequently the operation is done on traumatic cataracts, or on soft cataracts already discissioned. In either of these events the anterior chamber will contain lens matter. This is removed by opening the corneal incision by pressure on its posterior lip with a delicate spatula or spoon (Fig. 304), and the escape of the lens matter further facilitated by gentle counter-pressure and stroking at the point opposite the incision. This manœuvre is continued until the lens matter is evacuated as thoroughly as possible. The eye is then bathed with an antiseptic solution, atropine instilled, and a monocular dressing applied. It is rarely necessary to combine an iridectomy with this method. A modification of simple linear extraction consists in extraction of the lens, when sufficiently fluid, by suction. A curette with a hollow handle and partially covered bowl is introduced into the eye, and, by means of a rubber tube attached to the end of the handle, the operator draws the lens matter out by suction with his mouth. The same result is attained by means of a piston syringe, but this method is not much practised. Another modification consists in irrigating the anterior chamber, as will be described in treating of the extraction of hard cataract.

It occasionally happens that a patient over thirty years of age desires operation on a cataract which is either congenital or appeared during childhood. It should be treated as if it had developed but recently; that is, by extraction, as if it were a hard cataract, which it will have by this time become, if not manifestly hypermature.

Extraction of hard cataract may be performed in one of three principal ways: 1. After a preliminary iridectomy. 2. Combined with an iridectomy done as a step of the operation. 3. Without iridectomy (simple extraction).

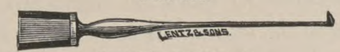
1. Extraction after Preliminary Iridectomy. This is probably the safest of all methods of extracting a hard cataract, but has the disadvantage of subjecting the patient to the danger and inconvenience of two operations involving opening of the eyeball. With much to be said in its favor on the score of safety, it is practised comparatively seldom. The iridectomy is to be done as described in Chapter VII., about six weeks before the extraction of the lens, which should not be undertaken until all signs of irritation following the first operation have disappeared. The technique of the extraction will be the same as to be presently described.

2. Extraction with iridectomy is the best operation for those of limited experience with the cataract operation. Its difficulties and dangers are fewer than those of the simple method and are easier to avoid. It is usually performed as follows:

Instruments. A Graefe knife (Fig. 298) (it is well to have two at hand), a stop speculum (Fig. 290), lid elevator, fixation forceps (Fig. 291), iris forceps (Fig. 295), iris scissors (Fig. 297), a blunt knife (Fig. 307) or scissors for enlarging the corneal wound, cystotome (Fig. 300), silver spatula (Fig. 304), spoon (Fig. 302), wire loop (Fig. 303), and blunt hook (Fig. 301). The lids may be separated

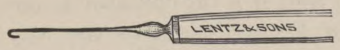
with a speculum or by a lid elevator under the upper lid, held by an assistant, who draws down the lower lid with the finger of the

FIG. 300.



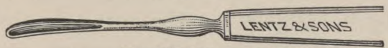
Graefe's cystotome.

FIG. 301.



Tyrell's blunt hook.

FIG. 302.

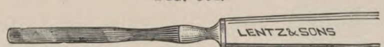


Daviel's lens scoop.

FIG 303.

Levis' wire loop.

FIG. 304.

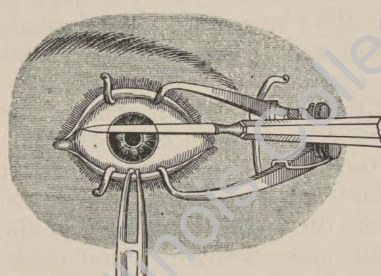


Spatula, shell or silver.

FIG. 305.

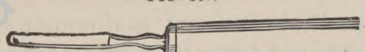
Wecker's iris scissors.

FIG. 306.



The incision in cataract extraction. Puncture and counter-puncture have been made. The section will pass in its whole extent exactly through the transparent margin of the cornea, the knife remaining in the same plane throughout. (Slightly modified from de Schweinitz, Diseases of the Eye, third edition.)

FIG. 307.



Desmarres' secondary knife, straight.

other hand. As it is often necessary to operate without skilled assistance, or without an assistant to whom the operator is accustomed, it is desirable to get in the habit of operating without an assistant. It is preferable to have no assistant rather than a poor one. The operator stands behind the patient, holding the cataract knife in the right hand and fixation forceps in the left for the right eye, and *vice versa* for the left. The eye is grasped below the cornea with fixation forceps, and if a lid elevator is used, these forceps may now serve to hold the lower lid out of the way. The incision is to be made in an upward direction. The eyeball is rotated downward and the point of the cataract knife entered just within the clear margin of the cornea, and at a point which is the temporal extremity of a line parallel to a tangent to the corneal summit, and dividing the cornea into two portions, the upper portion of which is one-third of the cornea. The knife enters the cornea at right angles to its surface, and as soon as the point is seen to have penetrated the cornea the handle is depressed until the point of the knife is directed straight across the anterior chamber from the site of the puncture, and the surface of the blade lies parallel to the surface of the iris. The knife is pushed forward, emerging from the cornea at a point (counter-puncture) diametrically opposite the puncture. (Fig. 306.) Up to this time the aqueous humor is preserved, and the point of the knife seen through it lies farther backward than it seems. The handle of the knife should be carried well backward toward the external canthus, or the points of puncture and counter-puncture will not correspond, and an irregular wound will be made. As soon as the point emerges from the eye the blade is pushed firmly inward and upward to its full length, and then drawn back, still pressing it upward. These two cuts will sometimes sever the cornea throughout its extent, but frequently a bridge remains requiring further to-and-fro movements of the knife to divide it. The knife should be held with its blade parallel to the plane of the iris throughout, and if this is done a smooth corneal incision results, with a small conjunctival flap at the apex of the corneal flap. The knife is now laid aside, and the fixation forceps turned over to an assistant or dispensed with if the patient is docile. The surgeon takes the iris forceps in his left hand between the thumb and index finger, and the iris scissors in the right hand. The closed iris forceps are gently insinuated into the wound, passed to the pupillary border of the iris opposite the centre of the wound, opened slightly, and made to grasp the iris at the pupillary border. They are then withdrawn, bringing with them a fold of the iris, which is drawn out of the wound as far as possible and cut off smooth with the cornea by means of the scissors. The fixation forceps may or may not now be reapplied. The cystotome is then introduced flatwise, carried to the centre of the pupillary space and turned, so that its point is directed toward the capsule. This is then freely cut, not torn, in such a manner as to give a sufficient opening for the lens to escape. Some operators make extensive

irregular incisions, others make incisions in the shape of a cross, the letter A, an inverted ʌ, or like the Greek letter π. Knapp makes a capsular incision parallel to the corneal incision (peripheral capsulotomy). The cystotome is turned and withdrawn carefully flatwise, and the surgeon takes the spoon in one hand and the wire loop in the other. The speculum or elevator may be removed at this time. Gentle pressure is made with one of these instruments just below the edge of the cornea, the pressure being first backward and then upward. The lens tilts and presents its edge in the corneal wound, which gapes from the pressure below. If it does not, it is made to do so by pressure on its upper lip with the instrument held in the other hand. By continuing the upward pressure the lens is slowly delivered from the eye (Fig. 308) and caught with one of the instruments held by the operator, and removed from the eye. If the speculum or elevator has not already been removed, it should be removed now, and the patient allowed to rest, with the assurance

Fig. 308.

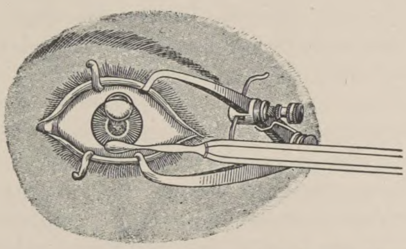


Fig. 309.

Fig. 308.—Delivery of the lens. The lens is presenting in the wound (capsulotomy has been performed). (De Schweinitz, Diseases of the Eye, third edition.)

Fig. 309.—The manner of applying the dressings after cataract extraction.

that the operation is over. In a few minutes he is told to open the eye, and the "toilet of the wound" is made. If any portions of the lens remain in the eye, they are coaxed toward the corneal wound by stroking the cornea with the back of the spoon, and delivered as the lens was by pressure on the cornea. It is best not to introduce any instrument into the anterior chamber to facilitate removal of this débris. A slender spatula is introduced, however, to ensure cleanliness of the wound, and to replace by gentle stroking the edges of the coloboma of the iris. All the clotted blood and other material is removed from the conjunctiva by moist pledgets of sterilized gauze or by the iris forceps, the small conjunctival flap smoothed out, a

drop of atropine instilled, and the dressings applied. These consist of squares of sterilized gauze two inches square and two layers thick, wet with 1:5000 mercuric chloride, then a small pad of sterilized absorbent cotton, both eyes being so covered, and the whole held in place by strips of isinglass or adhesive plaster applied as follows: The first strip passes over both dressings from temple to temple. A second strip passes from the cheek of the operated side upward and inward across the pad over the operated eye to the forehead over the sound eye. A third passes from the cheek of the sound side across the pad over that eye to the forehead over the operated eye. (Fig. 309.) Over this is placed a mask made of some light stiff material, to protect the eye from accidental injury. The hands of the patient may be fastened by a strip of bandage tied to each wrist and to the post at the foot of the bed on the same side, these strips being just short enough to prevent the patient touching the eyes with the hand.

The writer prefers the above dressing to the roller-bandage, because it is easier to apply, does not necessitate raising the patient's head, and is cool, clean, and comfortable. It makes sufficient pressure, and this cannot be increased by turning the head. The strings to the mask can be easily insinuated under the patient's head without moving the head, and should be of unequal length, so that the knots will be on the side of the face next to the operated eye. In this position the patient will not lie on them, and they are accessible.

3. Extraction without iridectomy, or simple extraction, is the operation of selection by many surgeons of large experience. It leaves the eye more natural looking—indeed, in some cases it cannot be told that an operation has been done on the eye. But it has never been shown that the visual results are better than by the combined method, while its performance requires more operative dexterity, and convalescence is apt to be interrupted by certain complications which do not obtain in the combined operation.

The same instruments are required as for the combined operation. The operation is performed in the same way up to the stage of making the iridectomy, except that it is important that the incision lies within the clear cornea. The cystotome is introduced immediately after the incision is completed, and the capsule cut as already described. The speculum may or may not be removed at this time, and the lens delivered as already described. Particles of lens matter are coaxed into the pupil and thence out through the wound, as in the other operation. The iris is then carefully replaced by stroking with a spatula. If it refuses to stay in position, but prolapses in spite of being reduced, a portion of it should be excised (iridectomy). The iris may be so bruised or torn by delivery of the lens that it is best to excise the bruised part. Otherwise, eserine ($\frac{1}{2}$ gr. to 1 oz.) may be instilled to keep the iris drawn inward from the wound, and the dressing applied.

Modifications of the Operation. The original cataract extraction was what is known as the flap operation, the incision comprising one-half of the corneal circumference. The length of this has been variously modified, so as to include any portion from one-fourth to one-half of the cornea. A 3 mm. flap or "short flap," as it is called, is popular. A very important modification was Graefe's peripheral linear incision. The knife was entered at a point 1 mm. from the corneal margin and 2 mm. below a tangent to its summit, and brought out at a similar point on the other side. The incision was about 10 mm. long, and but slightly curved. Iridectomy was always done. The operation is not popular, because the conjunctiva is cut freely and bleeds to an annoying degree, and the position of the wound favors loss of vitreous and cyclitis. Mention may be made of the downward incision, and of one lying in the cornea and near its centre. These are rarely used.

Instead of delivering the lens by pressure below with a spoon, the speculum may be removed and the pressure made with the lower lid.

The anterior chamber may be irrigated to remove fragments of the lens cortex which remain behind. This is done by specially constructed syringes having curved and flattened points made of gold. The best fluid is a 0.6 per cent. saline solution, which should be warm and, of course, sterile. Boric acid may be used, but stronger solutions will damage the cornea. This method is not much in vogue, as it is somewhat troublesome and not specially advantageous. A compromise between the simple and combined methods has been effected by some operators, as follows: instead of grasping the iris at its pupillary border with the iris forceps, withdrawing it, and making an iridectomy, a small fold of iris is picked up midway between the root of that membrane and its pupillary border, drawn to the wound, and cut off, so as to leave a small hole in the iris. This operation leaves a round pupil, and by affording a passage for the aqueous humor from the posterior to the anterior chamber near the corneal wound, is thought to be less likely to be followed by prolapse of the iris. In certain cases where prolapse of the vitreous is especially apt to occur, or as an extraordinary precaution against prolapse of the iris after the simple operation, Kalt has proposed the corneal stitch. It is of fine silk, and is inserted before making the corneal incision in the same manner as the Lembert intestinal suture is inserted. A fine rounded needle is used, and the central loop is left very long, so that it can be drawn out of the way of the instruments used during the operation. After the completion of the operation the ends of the suture are drawn taut and tied. A portion of the anterior capsule may be removed by specially devised forceps, or by iris forceps. This is desirable if the capsule is thickened. An attempt should be made to make a more or less circular cut with the cystotome if this procedure is contemplated.

Some operators omit the capsulotomy, and attempt to rupture the zonule of Zinn and remove the lens in its capsule. This is done by

pressure with the spoon at the margin of the cornea. It renders loss of vitreous more probable, but when successful leaves a perfectly clear pupil. It is not much in vogue.

Choice of Operation. The safest method is that of preliminary iridectomy, followed by removal of the lens after some weeks. The patient is subjected to the danger of two operations, but the operator will have gained the patient's confidence and some knowledge of his behavior and that of the eye. It should certainly be done if the other eye has been operated on unsuccessfully.

The choice between the two principal methods, with and without iridectomy, is a matter on which opinions differ. The occasional operator had better use the former method, since it is, on the whole, easier to perform, and the visual results are quite as good as by the simple method. It is well, at any rate, to make the corneal incision as above described, and do the iridectomy if delivery of the lens is not easy, or if the iris tends to prolapse. When the iris is rigid or in the least discolored, or if the patient is restless, or if the incision has been placed other than within the clear cornea, it is better to do the iridectomy at once, as the iris is likely to prolapse and necessitate its performance later, and prolong the healing.

An operator who is not ambidextrous should take his position in front and at the left side of the patient in operating on the left eye, and, inserting the knife with the right hand, cut upward—*i. e.*, away from himself. To avoid the necessity of assuming this position, angular knives (Fig. 299) have been devised, by which the incision is made by entering the cornea to its nasal side. They are seldom used.

Accidents during the Operation. The knife may be inserted upside down. It should be turned in the wound and the operation proceeded with, or the knife may be withdrawn and the operation postponed. As the aqueous is lost by withdrawing the knife and the iris and the cornea lie in contact, the knife cannot be reinserted.

The iris may fall over the edge of the knife. By raising the knife it may be disengaged; but if not, the incision should be completed, the fold of the iris being cut through. Then with the iris forceps, the cut portion of the iris is drawn out and the wound made as nearly an ordinary iridectomy as possible.

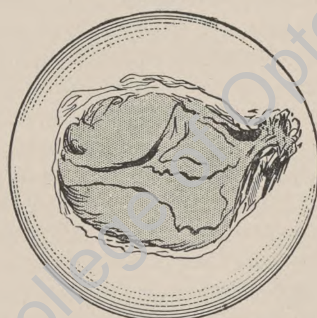
Loss of some of the vitreous humor is a common accident. It is rendered less apt to occur by removal of the speculum at the completion of the capsulotomy. If this is not done and a bead of vitreous presents in the wound at any stage of the operation, the speculum should at once be removed and the patient allowed to rest a moment. If the lens has not been extracted, the attempt to deliver it by pressure in the usual way would probably result in loss of the vitreous. Therefore, no pressure should be made on the ball, but the lens should be removed by the wire loop gently passed through the wound and under the lens. Loss of vitreous usually occurs after delivery of the lens. Fluidity of the vitreous or a sudden, voluntary squeezing of

the muscles around the eye, or an involuntary contraction of the recti muscles, may cause the loss of a considerable amount of vitreous. If this is not sufficient to cause collapse of the globe, it is usually not a serious matter and requires no treatment; but if the eyeball collapses, warm sterilized salt solution (0.6 per cent.) should be injected into the eye by a pipette introduced through the corneal wound, until the ball resumes its rotundity. This will generally prevent any permanent ill effect from the accident. If the wound is too small to allow the easy passage of the lens, it should be enlarged. This can be done by a blunt-pointed knife (Fig. 307) or fine blunt-pointed scissors, such as Stevens' tenotomy scissors. Under no circumstances should the lens be forced through a wound which is evidently too small.

The lens may be dislocated. This is usually done in the attempt at capsulotomy when the capsule is thick or tough and the suspensory ligament frail. The dislocation is generally backward. The lens should be caught by the wire loop passed through the wound, and gently drawn from the eye.

Hemorrhage into the eye may occur from the cut iris or from the rupture of a choroidal vessel due to lack of support to these tissues which opening of the eyeball entails. The former is usually a trivial matter, the hemorrhage ceasing spontaneously or being made to cease by compresses wet with hot antiseptic solutions being applied to the closed lids. Blood left in the anterior chamber at the termination of the operation may be expected to absorb in twenty-four to forty-eight hours. Hemorrhage from the choroid is fatal to the eye. The patient at any time after the completion of the corneal incision, or even several hours after the operation is finished, complains of severe aching pain in the eye, and there occurs gaping of the wound, then loss of vitreous, and a free flow of blood filling the ball and pouring from the wound. A compress and bandage should be applied, a hypodermic of morphine given, and the patient made to sit up. The bleeding will cease in from a few minutes to a few hours, but the eye is always irretrievably lost, and subsequently shrinks. (Fig. 310.) The pain may continue to such a degree as to justify enucleation. The accident is rare.

FIG. 310.



Specimen of choroidal hemorrhage following cataract extraction. The globe is filled with blood, and the retina and vitreous have been expelled. The dark line is the choroid, which has been everywhere torn loose (life size). (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

Sometimes after the cataract operation the patient will develop a maniacal condition, probably due to the combined mental effect of nervousness from operation, exclusion of light, and the lack of any-

thing to occupy his attention. It is treated by sedatives, allowing him to use the unoperated eye, and by having someone remain by his bed to talk to him and otherwise "keep him company." Patients accustomed to the free use of alcohol frequently develop delirium tremens after this operation, as after other surgical operations.

The After-treatment of Cataract Extraction. In a case which runs a normal course the first thing to be observed is closure of the wound and reformation of the anterior chamber. As long as patency of the wound permits the aqueous to flow away the anterior chamber is empty and the iris rests against the cornea. Closure of the wound is shown by accumulation of aqueous forcing the iris back to its normal position. Until this happens we must feel some apprehension of possible infection, and, in the simple operation, of prolapse of the iris. Closure of the wound usually occurs within twenty-four to thirty-six hours. Atropine may be used immediately after the operation in cases where iridectomy is done; but after the simple operation should not be used until the wound has closed, for fear of inducing prolapse of the iris. The eye should be dressed daily, and the condition of the lids observed for swelling or other indications of inflammation. The lids should be separated and the ball inspected, too, and in cases done without iridectomy the wound should be looked at to see if prolapse of the iris has occurred. As soon as the wound closes the patient may be allowed to sit up and the sound eye left uncovered. After forty-eight hours more the dressings may be left off and dark glasses or a shade substituted. Confinement to the room is necessary for at least a week, and atropine should be kept up, usually three times a day, until the eye is entirely free from redness. The eyes should not be used for reading or other near work before this time.

The immediate or early use of atropine after cataract extraction is justified by the frequency with which the operation is followed by at least a mild degree of iritis. We aim to secure dilatation of the pupil before this occurs, which is generally about the second or third day, or later. We observe injection of the ball, especially in the pericorneal zone, slight pain and tenderness, photophobia, and a tendency for the iris to adhere to portions of the capsule and any remaining fragments of lens. In favorable cases these symptoms disappear in about two weeks.

Patients complain bitterly of pain in the back when confined to the bed in the prone position for twelve hours or longer. This may be relieved by slipping a small pillow under the small of the back, or by turning the patient gently on the side away from the operated eye. Until the wound closes, only food which does not require chewing should be allowed, and the patient should be made to use the bedpan and urinal if possible rather than rise or sit up. The open method of treatment, or that of placing no dressing at all on the eye, proposed by Hjort, has not found followers.

Anomalies of Healing. Delayed closure of the wound may result from entanglement of a tag of capsule or other foreign matter in the

wound. If not closed in thirty-six hours, careful search should be made for such cause, and the particle removed with forceps. Frequently the wound heals slowly from a poor state of nutrition or from no apparent cause. Conjunctivitis with discharge may arise from the action of the occluding bandage. The dressings should be lightened or left off entirely, and the eye frequently irrigated with a boric acid solution. If the discharge does not cease, the lids should be everted and a solution of nitrate of silver (4 gr. to 1 oz.) applied to the conjunctiva. Even if the wound is open, this should be done, as the risk is less than that of allowing the discharge to continue. If the wound has closed, the danger of infection is lessened. Iritis occurs very frequently. It usually yields to atropine, but if severe may require rest in bed, the addition of cocaine, leeching, ice compresses, and antiphlogistic doses of mercury. Ice acts far better in these cases, which are traumatic, than heat.

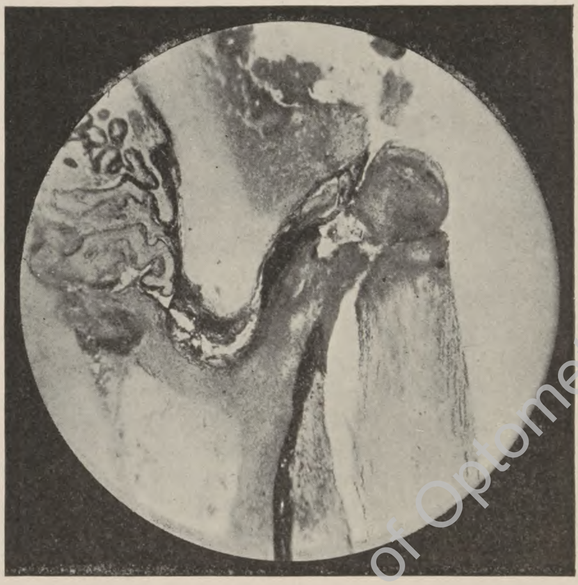
Iridocyclitis and iridochoroiditis are to be treated in the same way. They are much more serious than simple iritis, and may result in destruction of the eye by subsequent atrophy. In common with iritis, they tend to form exudations in the pupillary space, the membranous secondary cataract resulting being very tough and dense. In iridochoroiditis in particular we notice chemosis of the conjunctiva, and in all the inflammatory states which may follow the cataract operations the lids swell, especially at the inner canthus. In the absence of this sign and discharge we may feel reasonably sure of the absence of undue reaction.

Prolapse of the iris is the most common complication of the simple operation, and constitutes the greatest objection to it. It occurs in from 3 to 10 per cent. of cases. Its occurrence is often announced by a sharp pain, but as often by no unusual sensation. If discovered before inflammatory action has sealed it firmly to the cornea, the prolapse should be excised and the margins of the coloboma dressed back into the eye. It is hardly wise to replace the prolapse and try to hold it in place by eserine, though this is sometimes done. If the prolapsed portion is firmly sealed in the wound by inflammation, it may be incised, touched with the actual cautery, or left alone. It will eventually shrink and flatten, so as to leave no trace but a small pigmented spot in the wound; but as entanglement of the iris is apt to lead to iritis or iridocyclitis (Fig. 311), or form a path for infection, it should be excised when possible. Otherwise, to let it alone is preferable to incision or the use of the cautery.

Septic infection is a dreaded and usually fatal complication. It may arise in the wound or from the iris (suppurative iritis), or more rarely in the vitreous. The first is usually by infection from without after operation, the other two forms by operative infection. Suppuration of the wound is most common. It is announced by pain and marked inflammatory symptoms, and the wound is found to present swollen edges and a yellowish infiltration along its course. This may spread toward the corneal centre or around the cornea,

like a ring. The whole cornea becomes opaque, and usually sloughs off. The eye subsequently shrinks. Sometimes the suppuration is limited, and healing may occur with some remaining sight or a possibility of obtaining some by operation. The treatment should consist in thorough frequent cleansing of the eye with 1:10,000 mercuric chloride and the application of the actual cautery or pure carbolic acid to the line of the wound. These measures may be repeated. Subconjunctival injections of mercuric chloride may be tried, but are very painful and usually useless. But the course of the condition is nearly always unchecked by any treatment. If the sup-

Fig. 311.



Prolapse of iris after cataract extraction. The corneal lip of the wound is infiltrated and displaced. The lower part of the iris is drawn up by the exudate. Enucleation on the tenth day following the extraction. × 15. (Prepared by Dr. E. S. Thomson, in the laboratory of the Manhattan Eye and Ear Hospital.)

puration is from the iris, or if pus is seen within the anterior chamber, the wound should be opened and the anterior chamber irrigated with a boric acid solution. The introduction of powdered iodoform or rods made of iodoform and gelatin has been recently tried in this condition, and promises better results than any other treatment. If the infection is primarily in the vitreous, injections of mercuric chloride into that body may be tried.

Traumatic striped keratitis is the name given to a form of corneal inflammation manifested by parallel gray lines running in the corneal substance from the wound toward its centre. It is due to bruising

of the anterior flap by the lens, and is caused by a too small wound. It subsides in a few days.

Occlusion of the pupil by the iris is due to entanglement of the iris in the corneal wound. It may occur after either the simple or combined operation. The iris is stretched over the entire bottom of the anterior chamber, or a small pupillary opening may be left near the wound. The treatment is by iridotomy.

Cystoid Cicatrix. This is a bulging of a portion or all of the corneal wound, due to the cicatricial tissue yielding to intra-ocular pressure. The adjacent conjunctiva is sometimes involved. It is to be treated by a firm compression bandage worn for several weeks. This sometimes fails to relieve the condition, under which circumstances the site of the original wound may be exposed by turning up a small conjunctival flap, the leaking point found and touched with the galvanocautery.

Glaucoma sometimes develops after cataract extraction, probably being induced by the use of atropine in eyes predisposed to the disease. It is to be treated on the same principles as glaucoma occurring under other circumstances, but as a rule yields to the use of eserine and heat. If not, an iridectomy, sclerotomy, or even sympathectomy, may have to be done.

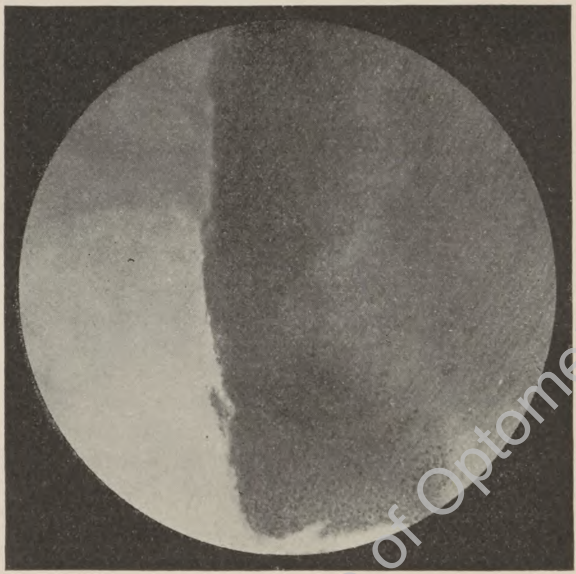
Secondary or After-cataracts. In a majority of cases there remains after the removal of the lens a membranous opacity, called secondary or after-cataract. This consists of the posterior capsule, with possibly some of the anterior capsule, and it may be thickened by the deposit on it of inflammatory exudate from the iris. Secondary cataract varies, therefore, in density from a filmy membrane which offers no obstacle to vision, to a dense, tough membrane completely abrogating useful vision. Provided the vision is not better than 20/50 and the reduction of vision is not manifestly due to some other cause, the membrane should be divided. The operation is known as capsulotomy. It should not be done until the eye is entirely free from the redness and inflammation following the primary operation.

Instruments. Speculum (Fig. 290), fixation forceps (Fig. 291), needle and knife needle (Fig. 292).

Treatment. The pupil is dilated with atropine, the eye anæsthetized, the lids separated by the speculum, and the eye grasped with fixation forceps. Artificial light is preferable, and should be concentrated on the field of operation by means of a reading-glass held by an assistant. The knife needle is entered near the corneal margin, and the membrane penetrated and cut through in such a manner as to leave an opening in the centre of the pupil. This is very well accomplished by making the incision in the form of an inverted Λ, the tongue-shaped flap being pushed backward by the needle before it is withdrawn from the eye. If too rigid to remain bent out of the way, it may be partially divided across its base by a third incision. The cutting edge of the knife needle should be extremely sharp, and the membrane

cut through by rapid short sawing strokes. This is done to avoid traction of the ciliary body, which would probably cause cyclitis. If the membrane is too tough to cut readily, it should be transfixed first by a needle, then the knife needle entered through the opposite side of the cornea, and thrust through the membrane near the needle. It is made to cut away from this point, the needle acting as the point of resistance, to protect the ciliary body, or two needles may be entered on opposite sides of the cornea and made to pierce the membrane near the centre. By approximating the handles of the points separate, the cornea at the site of penetration acts as a fulcrum, and the membrane is torn.

FIG. 312.



Leucocytic infiltration of lens. From a case of iridocyclitis following a wound in the ciliary region. $\times$ 100. (Prepared by Dr. E. S. THOMSON, in the laboratory of the Manhattan Eye and Ear Hospital.)

When the pupil is small and undilatable, Noyes proposed to enter a thin cataract knife through the sclerotic behind the ciliary body, and to transfix and cut the membrane from behind.

If the pupil is occluded by drawing the iris over it, this should be dealt with by iridotomy. The incision may be made with the knife needle or with Wecker's scissors (Fig. 305), introduced after making a sufficiently large corneal wound with a broad needle or keratome. The scissors are introduced closed, opened in the anterior chamber, and one blade made to penetrate the iris. The iris lying between the blades is then divided, usually in a V or cross-shape.

After the completion of capsulotomy or iridotomy atropine is instilled and the eye covered with a dressing. The patient should be kept in bed for twenty-four hours, and the least sign of iritis should be promptly met by leeching and ice compresses. Otherwise, inflammatory products will soon fill the opening and nullify the effect of the operation.

Accidents. The accidents most likely to happen are infection, glaucoma, intense inflammatory reaction, and detachment of the retina. The treatment of these conditions is given elsewhere.

Aphakia. Aphakia is the name given to absence of the lens, and is the condition that exists after a cataract has been removed. Its principal characteristic is an increase in the refraction of the eye by the dioptric value of the lens, usually 10 D. or 12 D., loss of all power of accommodation, and in cases of cataract extraction by the development of corneal astigmatism at right angles to the direction of the corneal incision. This astigmatism is great at first, usually from 3 D. to 5 D., but as cicatrization progresses it diminishes, usually to 1 D. Some patients possess a sort of pseudo-accommodation, which is generally performed by squinting or partially closing the lids. The refraction should be worked out by the same means as used in estimating refraction under other circumstances, and reading-glasses of 3.50 D. or 4 D. stronger than the distance correction also given. Bifocal lenses, or, if only one eye is useful, reversible frames, are to be given. The strength of the correction for near work must be made to accord with the distance at which the patient will use his eyes most.

Inflammation and **new-growths** of the lens do not occur, although the lens may be the seat of infiltration of leucocytes in cyclitis. (Fig. 312.)

CHAPTER XI.

GLAUCOMA.

By E. TREACHER COLLINS, F.R.C.S. Eng.

The term "glaucoma" is derived from the Greek word γλαῦκος, signifying sea-green. It was used by Hippocrates, and was applied originally to affections of the eye in which a green or greenish-gray reflex was obtained from the pupil. At different times the disease has been regarded as an affection of the crystalline lens, an affection of the vitreous humor, and an effusion between the retina and choroid. It was not until the discovery of the ophthalmoscope in 1851 that these several theories respecting it were found to be untenable.

Mackenzie, of Glasgow, in 1830, first drew attention to the increased tension of the eye in glaucoma, which is now known to be its essential factor. As was pointed out first by von Graefe, all the other symptoms can be explained as the result of increased tension.

Glaucoma may now be defined as increased tension of the eye, the result of derangement in the circulation of the intra-ocular fluid.

A green reflex from the pupil is not always present in glaucoma, and it may be met with in other conditions in which there is no increase of tension.

A derangement of the circulation of the intra-ocular fluid causing increase of tension may occur in a variety of ways. It may occur in an eye which in other respects is apparently healthy, or it may be the result of some obvious precedent disease. In the former case the glaucoma is termed *primary*, and in the latter *secondary*.

The Mechanism for the Maintenance of Normal Ocular Tension. There are three sorts of fluid within the eyeball, variable in amount: blood in the bloodvessels, lymph in the lymphatic spaces of the uveal tract and the perivascular lymphatic channels, and the intra-ocular fluid in the aqueous and vitreous chambers. The amount of blood in the intra-ocular bloodvessels is subject to constant variation from many causes, such as alterations in the blood pressure, changes in the shape of the iris and ciliary body, and varying amount of pressure from the surrounding muscles.

The lymph is derived from the bloodvessels, and its amount is dependent on the blood pressure.

The intra-ocular fluid contained in the aqueous and vitreous chambers is of practically the same consistency. Its composition is estimated as 99 per cent. water, 1 per cent. salts and extractives, together with a trace of albumin.

In the vitreous this fluid is lodged in a network of fibres much like water in a sponge, and is surrounded by a hyaloid membrane. It is this arrangement which gives to the vitreous humor its gelatinous consistency.

The intra-ocular fluid is a secretion, and not a mere exudation from the bloodvessels. If it were an exudation, it would contain a large quantity of albumin, like lymph.

There is considerable experimental and clinical evidence to show that glaucoma is produced by the secretive action of the epithelium covering the ciliary body. The folds of the ciliary process provide a comparatively large epithelial covered surface overlying a dense plexus of bloodvessels. There are, moreover, on the pigmented layer numerous little tubular recesses, presumably glands, concerned to some extent in the elaboration of secretion.

Experimentally it has been found that after excision of the iris and ciliary body from the eye of a rabbit, the secretion of the aqueous humor is arrested and the vitreous shrinks. Also, that subcutaneously injected fluids make their appearance in the eye first at the ciliary body, and thence spread to the vitreous, and through the pupil to the anterior chamber.

Clinically, we find that when the pupil becomes closed by a complete ring of posterior synechiæ the aqueous humor accumulates behind the iris, bowing it forward. Further, that all the vascular structures within the eye, other than the ciliary body, may be absent, or have their vessels occluded, without alteration in the amount of the intra-ocular fluid or the tension of the eye being noted; while destructive processes involving the ciliary body cause shrinking of the globe. Thus, the tension of the eye and the intra-ocular secretion have been found unaltered when the following conditions were present: congenital and traumatic aniridia, embolism of the central artery of the retina, congenital absence of the choroid. The tension has also been known to remain increased, where all the bloodvessels supplying the retina and choroid have been cut through, after the operation of optico-ciliary-neurotomy performed for absolute glaucoma.

Some of the secretion from the ciliary body passes directly forward between the iris and lens into the anterior chamber. The main exit for fluid from the anterior chamber is, as first proved by Leber, at its angle. It passes through the spaces of Fontana in the ligamentum pectinatum, into the canal of Schlemm by a process of filtration, and from there into the anterior ciliary veins. A certain amount, Nuel[1] has shown, also escapes through the iris, entering the openings on its anterior surface, which are situated mostly near its ciliary and pupillary margins, then into the iritic veins by filtration through their walls.

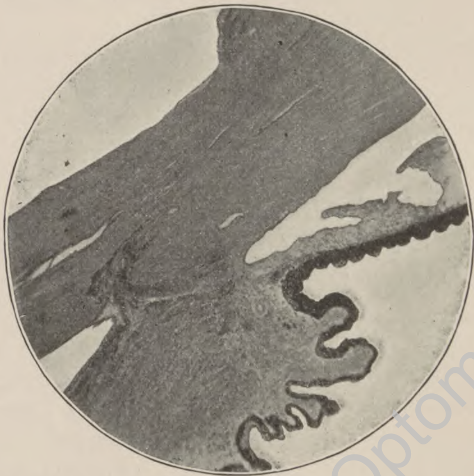
A part of the secretion of the ciliary body passes to the vitreous humor. From the vitreous a small amount of fluid may escape along

[1] Arch. d'Ophtalmol., April, 1900.

the lymphatics around the central retinal vessels in the optic nerve, or by filtration into the larger vessels themselves; most, however, after permeating the anterior hyaloid membrane and suspensory ligament, passes through the circumlental space and pupil into the anterior chamber.

In a hollow sphere distended with fluid the amount of pressure on the walls is equal at all points. In the eye, which is divided into two chambers, the aqueous and vitreous, by a diaphragm consisting of the lens and its suspensory ligament, it is conceivable that the pressure in one or the other might be greater. In the normal condition this is not the case. The pressure in the vitreous chamber

FIG. 313.



The angle of the anterior chamber in a healthy eye, showing the canal of Schlemm, the ligamentum pectinatum, and lymphatic crypts at the periphery of the iris.

and in the anterior chamber, measured experimentally by a manometer, has been found to be equal in each to a column of mercury about 28 mm. in height. The equality of pressure is maintained by the possible free escape of fluid from the vitreous into the anterior chamber, and free escape of fluid from the anterior chamber out of the eye.

In spite of changes which are constantly occurring to alter the amount of blood in the intra-ocular bloodvessels, in the normal condition the tension of the eye, as estimated by the finger pressure, remains practically uniform.

By a more active secretion of the ciliary body, or by a more rapid escape of fluid, the eye has the power of adapting itself so as to maintain an equable amount of pressure upon the structures contained within.

It would seem natural to suppose that this regulating power which maintains a uniform degree of tension is the result of nervous in-

fluences. Our knowledge, however, of the influences of the nervous system on the tension of the eye is at present far from complete, and, in some respects, contradictory.

We should like to know if the eye, cut off from all influences proceeding to it from the cerebro-spinal or sympathetic nervous system, is capable of maintaining normal tension.

The results of the operation of optico-ciliary-neurotomy offer us some evidence on this point. When performed on an injured eye as a prophylactic against sympathetic ophthalmitis, the tension frequently becomes diminished, and, in some cases, this diminution of tension appears to be the direct result of the operation, and not caused by the affection of the eye for which it was performed. When performed on eyes with absolute glaucoma, where presumably the channels of exit of fluid from the eye are closed, the tension remains increased.

Nervous influences may proceed to the intra-ocular structures either through the trigeminus or through the sympathetic nerves.

The experiments of dividing or irritating the trigeminus in animals have not produced uniform results. Some observers have found its division result in diminished tension, and its irritation in increased tension (Donders), while others have concluded that its division or stimulation had no influence on ocular tension (Wegner).

The operation of removal of the Gasserian ganglion has now been performed a number of times on man for the relief of neuralgia, but in the description of such cases no reference is made to its effect on ocular tension. It is possible that, unless specially looked for, some slight variations in tension may have been overlooked.

In the affection known as herpes ophthalmicus, which is due to a lesion of the Gasserian ganglion, diminution of ocular tension is sometimes noted.

Experiments on animals have shown that section of the cervical sympathetic causes diminution of ocular tension, while irritation of it occasions a transient increase of tension (Wegner, Adamuk).

Removal of the superior cervical ganglion has been performed in man, in patients who had no ocular affection, without any alteration in the ocular tension being appreciable (F. F. Burghard[1]).

Removal of the superior cervical ganglion in patients with primary glaucoma sometimes reduces the tension (Jonnesco[2]). In some cases of paralysis of the cervical sympathetic from injury or pressure, a slight diminution of ocular tension has been noted.

In cases where symptoms of stimulation of the cervical sympathetic are present, as in Graves' disease, glaucoma has not been proved to be of unusually frequent occurrence.

Stimulation or removal of the superior cervical ganglion causes several changes in and about the eye, which it is conceivable would

British Medical Journal, October 20, 1900, p. 1175.
Wiener klin. Wochenschrift, May 4, 1899.

tend to influence the intra-ocular pressure. Thus its stimulation causes:

1. *Dilatation of the pupil,* which might impede the exit of fluid from the eye by narrowing the orifices of the lymphatic spaces on the anterior surface of the iris, or by approximating the root of the iris to the back of the cornea in the region of the spaces of Fontana.

2. *Increased blood pressure,* which diminishes the amount of blood in the eye, and so lessens the amount of its contents, but also probably increases the amount of lymph in the intra-ocular lymphatics.

3. *Increased secretion from the ciliary body,* which tends to increase the contents of the eye.

4. *Contraction of the unstriated muscle fibres of Müller,* which by compression of the efferent veins coming from the eye delays the exit of blood from it.

Removal of the ganglion produces just the reverse effect: it contracts the pupil, lowers blood pressure, diminishes secretion, and allows of relaxation of the muscle fibres of Müller.

Another factor calling for consideration in the maintenance of normal intra-ocular tension is variation in the composition of the intra-ocular fluid. It has to escape from the eye, as already mentioned, by a process of filtration. Containing, as it does in the normal state, but a trace of albumin, this filtration may readily take place. Should, however, the anterior chamber be punctured and the aqueous humor allowed to escape, the fresh aqueous humor, which is formed much more rapidly than under the usual conditions, is found to contain a considerable quantity of albumin, and will consequently take much longer to filter out of the eye.

The Effects of Increased Tension on the Various Structures of the Eye and Their Respective Functions.

The maintenance of the normal amount of intra-ocular pressure is most essential for the regular performance of the functions of the different structures composing the eyeball. We shall now proceed to describe the changes which are produced in an eye as a result of a disturbance in the intra-ocular pressure, resulting in increased tension.

Sclerotic and Conjunctiva. A sudden onset of increased tension so disturbs the intra-ocular blood circulation as to cause for a time a general congestion of the ciliary bloodvessels in the sclerotic, and often also of those of the conjunctiva. In the most acute cases this congestion is accompanied by œdema of the conjunctiva (chemosis), and sometimes even œdema of the eyelids. The vessels being mostly engorged with venous blood, the injection has a characteristic dusky hue. The main exit of blood from the uveal tract is by the venæ vorticosæ; the channels in the sclerotic through which these pass run very obliquely, and when the sclerotic is stretched, as it is in glaucoma, they easily become closed. The result of such obstruction is to cause considerable enlargement of the anterior ciliary veins, which

normally give exit to only a small portion of the venous blood from the ciliary body, and which perforate the sclerotic more at a right angle than the venæ vorticosæ.

Increase of tension of some duration will cause hypertrophy of the anterior ciliary arteries from the increased obstruction to the entrance of blood into the eye.

When the onset of increased tension comes on gradually, instead of suddenly, an adaptation of the intra-ocular blood circulation to the altered conditions is rendered possible, and the violent disturbance resulting in congestion and œdema does not take place. Indeed, some cases of primary glaucoma develop so slowly that scarcely any alteration in the state of the ciliary bloodvessels is to be observed, or at most slight enlargement of the anterior perforating vessels.

The effects of increased tension on the sclerotic vary very much according to the age of the patient. In early life the sclerotic is an elastic structure; as life advances, it becomes tougher and less expansible. Consequently, if increase of tension is met with in infancy or early childhood, the sclerotic will give and the whole globe become enlarged.

After adolescence, the sclerotic being hard and unyielding, little alteration in the shape of the globe is met with as the result of glaucoma; usually there is slight distention in the spaces between the recti muscles, so that the globe becomes somewhat square in shape.

Should there have been, previously to the onset of the glaucoma, some weakening or thinning of the walls of the globe, as from a patch of choroiditis, then, when the tension is increased, that spot is likely to give and become staphylomatous.

Cornea. As the result of increase of tension, some interference in the circulation of the lymph streams in the cornea may occur. This is especially liable to take place where the onset of tension is sudden. Its effect is to cause œdema at the anterior part; the spaces between the anterior lamellæ of fibrous tissue are found enlarged and filled with albuminous fluid. Spaces are also met with between the surface epithelium and the anterior limiting membrane, between the epithelial cells themselves, and in the channels in the anterior limiting membrane through which the nerve fibres pass to the epithelium. Clinically, this œdema gives rise to a superficial haziness of the cornea, which rapidly disappears when the tension is lowered.

In cases where the œdema has been present for a long time, small vesicles may form on the surface of the cornea; in some cases a new formation of fibrous tissue beneath the epithelium and external to the anterior limiting membrane takes place.

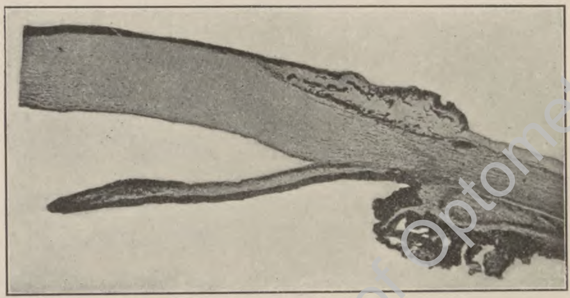
Anæsthesia of the cornea is a common accompaniment of glaucoma, and has been attributed to compression of the nerve fibres going to the epithelium by the fluid in the lymph spaces around them, as they pass forward through channels in the anterior limiting membrane. It may also be due to compression of the long ciliary nerve, from which

the corneal nerves are derived, against the hard, unyielding sclerotic, as they pass forward on the outer surface of the choroid.

The haziness of the cornea when present, apart from other causes, tends to make vision misty. It is also the cause of another very characteristic symptom of glaucoma, viz.: the appearance in the dark of halos of rainbow colors around lights, the red color always being the outermost.

A precisely similar appearance of halos of rainbow colors around lights is produced by dropping a solution of the alkaloid erythrophlæine, obtained from an African arrow poison, into the eye.[1] It causes also a slight steaminess of the surface of the cornea, slight anæsthesia, and some blurring of vision, but does not increase the tension. One specimen dilated the pupil and another contracted it; with both the halos were seen. Evidently then they are not the result of pressure on the retina or any alteration in the pupil. Halos may also be produced by the instillation of a drop of erythrophlæine in the eye of a patient who has undergone extraction of cataract, which excludes the lens from any participation in their production. We are led irresistibly to conclude that they are the result of the slight haze in the cornea.

FIG. 314.



The angle of the anterior chamber in a case of primary glaucoma, showing closure of the filtration area at the periphery of the cornea, by apposition with it of the root of the iris.

In primary glaucoma which occurs late in life no appreciable alteration in the shape or size of the cornea occurs. When, however, increase of tension is met with in infancy or early life, the cornea, like the sclerotic, being still very elastic, enlarges and becomes globular in shape. The condition is spoken of as keratoglobus or buphthalmos.

Anterior Chamber. The condition of the anterior chamber in glaucoma varies with the position at which the primary obstruction in the circulation of the intra-ocular fluid takes place. In primary glaucoma it is shallow; as will be shown later, this is due to an increase of tension occurring first in the vitreous chamber, and the lens with its suspensory ligament being forced forward. A continued shallowing of

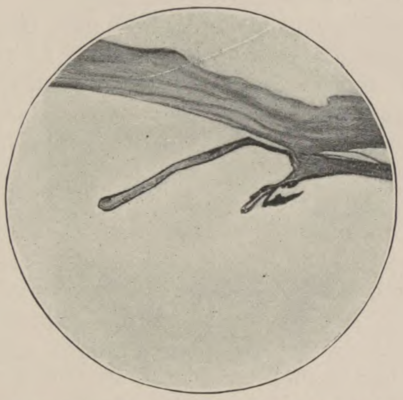
[1] Ophthalmic Review, 1890, vol. ix. p. 196.

the anterior chamber, and pressure of the ciliary processes against the root of the iris, result in contact of the latter with the back of the cornea and a narrowing of the angle of the chamber.

In some cases of secondary glaucoma and in cases of congenital glaucoma the primary obstruction to the circulation of the intraocular fluid is at the angle of the anterior chamber, where it gains exit from the eye; the anterior chamber then becomes deepened.

Iris. Pressure of the root of the iris against the back of the cornea leads to compression of both its bloodvessels and nerves. If the increased tension is sudden in onset, the compression at first causes œdema and venous engorgement, which make the iris appear altered in color. Later on, its vessels become empty and its stroma atrophies and shrinks. The pigment epithelium on the posterior surface of the

Fig. 315.



The iris and ciliary body from a case of glaucoma of long standing, showing marked ectropion of the pigment epithelium at the pupillary margin. The angle of the anterior chamber was closed by apposition of the root of the iris to the back of the cornea; in the preparation of the specimen they have become slightly separated. There is an abrupt bend in the iris where it ceased to be in contact with the cornea. The stroma of the iris is much atrophied, so that it appears very thin.

iris is unaffected by the atrophy. It normally ends at the pupillary margin; but in cases of glaucoma of long standing, by the shrinking of the stroma, the pigment epithelium becomes drawn around onto the anterior surface, a condition which is spoken of as ectropion of the pigment epithelium. Clinically it is seen as a dark pigmented area on the surface of the iris at the pupillary margin, usually extending more in one direction than another. It is most marked where the iris has become most atrophied and where the dilatation of the pupil is widest.

The pressure of the nerves against the back of the cornea in acute cases of glaucoma paralyzes the iritic muscles, and the pupil becomes inactive and semidilated. If the tension is relieved before atrophy has set in, its activity returns. In long-standing cases of glaucoma permanent dilatation of the pupil may be brought about through

atrophy of the sphincter muscle and shrinking of the stroma. The amount of dilatation is sometimes not equal in all directions, so that the pupil is often oval or irregularly circular, and it may be displaced away from the centre.

When the cornea and root of the iris have been in apposition for only a short while, their separation is easily effected. After a time, however, cell exudation takes place and they become adherent, and in long-standing cases of glaucoma most intimately adherent.

In chronic cases of glaucoma, where the onset of tension is gradual, and there has been time for compensatory changes to take place in vessels and nerves, the dilatation of the pupil and atrophy of the iris may be absent.

Ciliary Body. Increase of tension early causes disturbance in the accommodative action of the ciliary muscle, due probably to compression of the ciliary nerves against the sclerotic. It manifests itself by the apparent rapid advance of presbyopia, the patient requiring stronger and stronger glasses for near work. In the early stages of primary glaucoma, more especially in acute cases, the ciliary processes are swollen and œdematous, their veins are engorged, and they press forward against the root of the iris. After increase of tension has been established for some time, they become atrophied and shrink, as also does the ciliary muscle, so that in cases of glaucoma of long standing they are no longer in contact with the back of the iris, and a considerable space is left between them and the margin of the lens. The ciliary body receives an extensive nerve supply, from both the long and short ciliary nerves, which perforate the sclerotic posteriorly and pass forward in the lamina suprachoroidea, until they break up into a net-work of fine branches, which is known as the ciliary plexus. It is the sudden onset of pressure of this plexus against the unyielding sclerotic which is the cause of the excessive pain of acute glaucoma —pain which is not confined to the eye, but referred also to other parts supplied by the fifth nerve, especially those receiving branches from its first division. Certain reflex disturbances may also be set up, which will be referred to in speaking of the different clinical types of glaucoma.

When increased tension comes on gradually and is not very intense, the nerves, like the bloodvessels, have the power of adapting themselves to the changed conditions, and in the majority of chronic cases of glaucoma no pain is experienced by the patient from first to last.

Choroid. The effect of increased tension on the choroid in acute cases of glaucoma, as on the other portions of the uveal tract, the iris and ciliary body, is first to produce a condition of venous congestion and œdema; later on, emptying of its vessels and atrophy. In chronic glaucoma, on the other hand, no sudden disturbance of the circulation is set up, but the compression of the choroid against the sclerotic tends to empty the blood out of the capillaries. The fundus ophthalmoscopically in such cases is seen to lose its uniform red hue,

and to present a tessellated appearance, due to exposure of the network of larger vessels in its outer layers.

Atrophy of the choroid, the result of increased tension, is most marked at the parts where it has the firmest attachments with the structures external to it, viz.: around the optic disk and at the seats of exit of the vortex veins. When increase of tension has existed for some time, the optic disk is usually seen to be encircled by a yellowish-white ring, which is due to the atrophied choroid allowing the sclerotic to be exposed to view.

Lens. The displacement forward of the lens in primary glaucoma tends, if the eye is emmetropic, to make it myopic, as do also stretching of the walls and expansion of the globe in the antero-posterior axis. A drag on the suspensory ligament from displacement forward of the lens or expansion of the globe in the ciliary region would, on the other hand, lessen its refractive power, and tend to make an emmetropic eye hypermetropic.

In glaucoma in the adult hardly any expansion of the globe takes place, but it is stated that during attacks of glaucoma the refraction usually is increased.

In glaucoma in early life, where considerable enlargement of the globe in all its meridians is met with, the lengthening of the globe is compensated for in part by the flattening of the lens, and the amount of myopia met with is not as much as might otherwise have been expected.

In glaucoma of long standing the nutrition of the lens sometimes suffers, and it becomes cataractous. The opacity of the lens occasioned by glaucoma usually presents a bluish metallic lustre.

Aqueous and Vitreous Humors. Seeing, as already stated, that in acute glaucoma there are at first venous congestion and œdema of the iris and ciliary body, it might seem natural to suppose that there would be serous exudation into the aqueous and vitreous chambers, and that the intra-ocular fluid would be more albuminous than normal. Whether this is so or not, has yet to be determined definitely.

The haze of the cornea in acute glaucoma prevents it being seen how much the obscuration of the fundus may be due to want of clearness in the other media. That part of the increased brilliancy in the iris which is observed immediately after opening the anterior chamber in operations for acute glaucoma is due to escape of imperfectly clear aqueous humor, there can be little doubt.

The greenish-gray reflex from the pupil sometimes seen in glaucoma is probably in part due to an increased serosity of the media.

Retina. The immediate effect of increased tension on the retinal bloodvessels is to obstruct both the entrance of blood by the arteries and its exit by the veins. Consequently the latter become enlarged and the former smaller than normal. The intra-ocular pressure and the pressure of the blood in the retinal vessels are so balanced, under normal conditions, that no pulsation is to be observed in the retinal arteries. When the intra-ocular pressure is much increased, or the

arterial pressure much diminished, this balance is disturbed; blood then can force its way into the retinal arteries only during contraction of the heart, and pulsation in them becomes visible in the vicinity of the optic disk. If the increased tension in a case of glaucoma is not sufficient to give rise to pulsation of the retinal arteries, it may readily be elicited by slight pressure on the globe with the finger. Under normal conditions a considerable amount of pressure on the globe is required to produce pulsation.

Pulsation of the retinal arteries has been observed in cases of aortic regurgitation and of syncope, without increased tension of the eye and due to diminished blood pressure.

As the result of long-continued increased tension the walls of the retinal arteries become sclerosed. Hemorrhages into the retina from rupture of the small vessels are met with frequently in glaucoma.

A disturbance of the function of the retina, as the result of increased tension, may be due either to diminished blood supply or to atrophy of its nerve fibres.

Loss of vision due solely to the first cause is recoverable; that due to the second is permanent.

If the tension of a healthy eye be increased by pressure from without, as with the finger upon the eyelid, vision may be completely abolished, it disappearing last in the region of the macula. This may be attributed to arrest of the circulation in the retinal vessels, and possibly also in the choroidal capillaries from which the outer layers of the retina receive their nutrient supply. Directly the pressure is removed, the circulation is re-established and vision returns.

In the same way in acute glaucoma, vision may, in the course of a few hours, be reduced to mere perception of light or completely abolished. If normal tension is re-established before sufficient time has elapsed for organic changes in the nervous tissue to set in, vision will be restored.

The branches of the retinal artery which go to the periphery of the retina on the temporal side have a longer course to pursue than those distributed to other parts, because the point of entrance of the optic nerve into the eye is situated to the nasal side of the middle line. It is the capillaries, therefore, from the temporal branch which are affected first by any increase of tension.

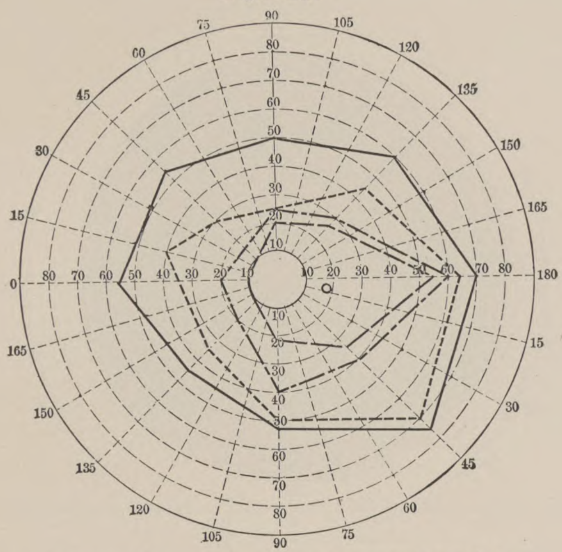
The nerve fibres destined for the periphery of the retina, which lie in the outer portions of the optic nerve, are more liable to be exposed to pressure against the sclerotic as they enter the eye, than those destined for the central regions.

These two anatomical facts serve to explain the manner in which vision fails in cases of glaucoma. The process begins at the periphery, producing a contraction of the field of vision. This contraction usually is noted first on the nasal side. As the case progresses, the field gradually becomes reduced to a more or less oval-shaped area, extending chiefly to the outer side of the fixation point. Ultimately the fixation point becomes involved, a small area in the field to its

outer side being left until the last. In some chronic cases of glaucoma perfect central vision may be retained with extreme contraction

FIG. 316.

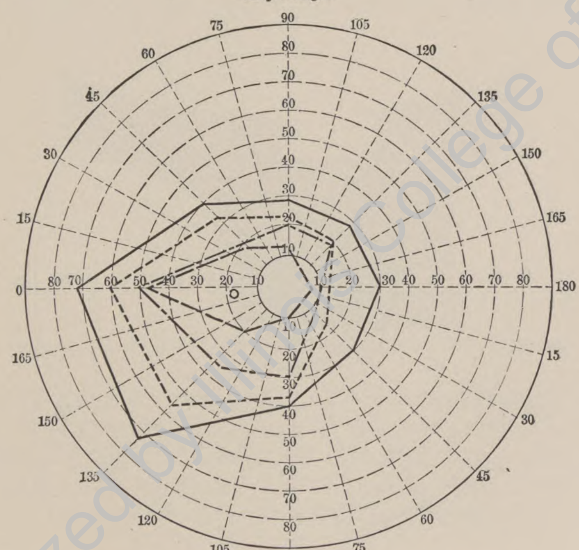
Right Eye



Concentric contraction as seen in glaucoma simplex.

FIG. 317.

Left Eye

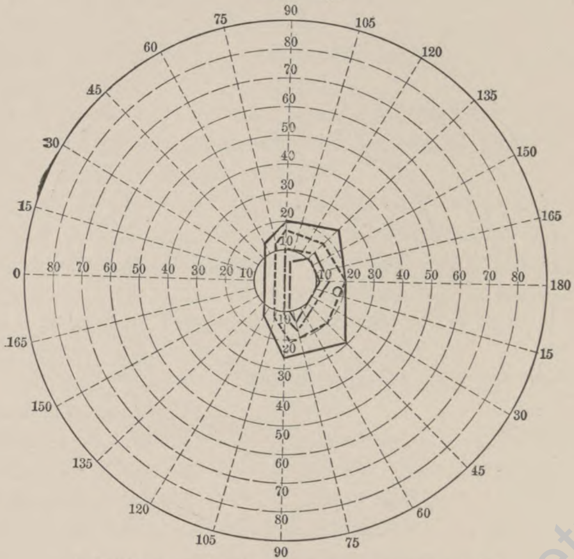


Concentric contraction as seen in glaucoma simplex.

of the field. In other cases, in association with a contracted field, some loss in the acuity of central vision is met with.

FIG. 318.

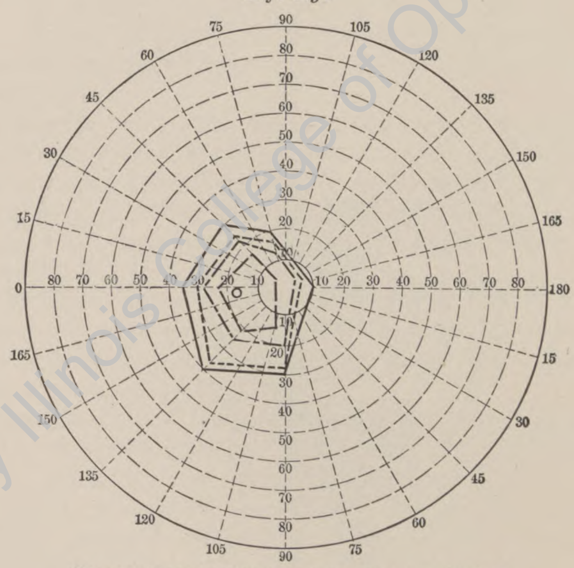
Right Eye



Concentric contraction as seen in glaucoma simplex.

FIG. 319.

Left Eye

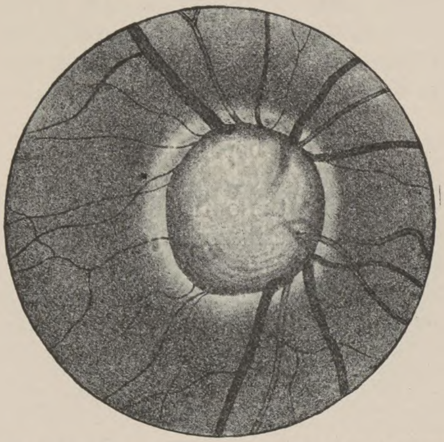


Concentric contraction as seen in glaucoma simplex.

Though the above is the most typical way for the field of vision to become affected in glaucoma, cases occur where it is contracted concentrically, or where there is a central or paracentral scotoma.

When the field is tested by an object which subtends a smaller visual angle than employed with the ordinary perimeters (as in the method

FIG. 320.



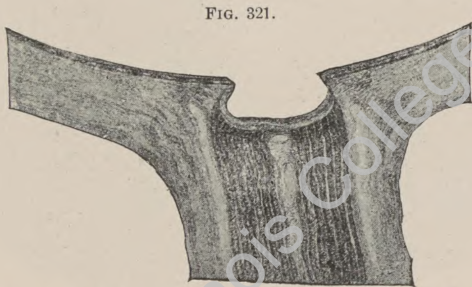
Ophthalmoscopic appearance of the optic disk in absolute glaucoma. (JAEGER.)

suggested by Bjerrum[1]), it is found that in glaucoma, whatever be the situation of the defect in the field, it always starts from the blind spot.

The fields for color usually fail proportionately to the field for white and to one another.

In eyes blinded by glaucoma there are found some atrophy of the

FIG. 321.



Section of optic nerve head of case depicted in Fig. 320. (JAEGER.)

nervous elements of the retina and some increase of the fibrous tissue elements. Small cystic spaces in the anterior portion of the retina, in the vicinity of the ora serrata, are met with very commonly.

Optic Nerve. The delayed exit of venous blood from the retinal vessels, and consequent venous congestion, probably give rise to

[1] Nordisk Ophthal. Tidsskrift, ii., 3, and Ophthalmic Review, 1890, vol. ix. p. 104.

some œdematous swelling of the optic papilla in many cases of acute glaucoma. By the time a clear view of the details of the fundus can be obtained, this swelling has given way to cupping and atrophy. Several observers have, however, described seeing appearances like optic neuritis as an initial symptom in glaucoma.

The position where the nerve fibres enter the globe is a weak spot in its walls. There, instead of having the three coats—sclerotic, choroid, and retina—there are only the fibres of the optic nerve and the lamina cribrosa. The latter does not represent more than half the thickness of the sclerotic, and is composed mainly of yellow elastic tissue.

When the tension of the eye becomes increased, this weak spot soon begins to give and bulge outward.

The sclerotic surrounding the optic disk is very thick and unyielding, so that, as the lamina cribrosa becomes curved backward, the nerve fibres become compressed against the tough resistant edge of the sclerotic at its margin, and consequently atrophy. When they become atrophied down to the lamina cribrosa, instead of, as in the healthy condition, there being an elevation in the region of the optic disk (the optic papilla), a depression is formed.

FIG. 322.

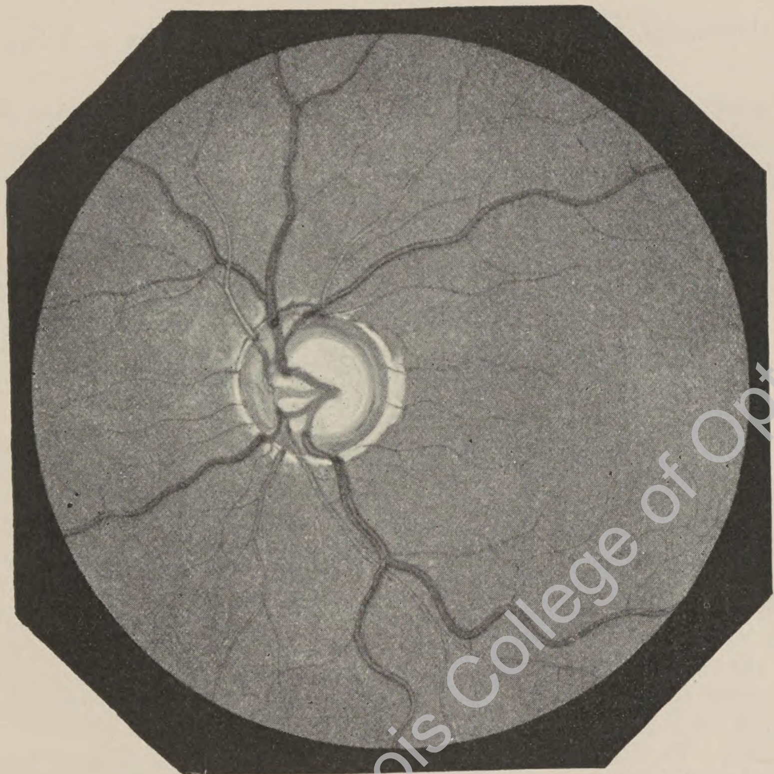
Cupping of the optic disk as the result of glaucoma. Besides depression backward of the lamina cribrosa there has been some lateral expansion, so that one side of the cup has become somewhat excavated. In the preparation of the specimen the retina has become displaced forward from contact with the choroid.

The cupping of the optic disk in glaucoma is then the result of two causes: depression backward of the lamina cribrosa and atrophy of the nerve fibres down to it.

As the depression backward of the lamina cribrosa increases, the sides of the cup tend to become quite steep; they may become expanded laterally at the posterior part, so that on section it presents a flask-shaped outline.

Ophthalmoscopically, a cupped condition of the optic nerve is recognized, with the indirect method of examination, by the parallax which is produced on movement of the lens. The bottom of the cup and the surrounding fundus seem to move at different rates, the former more slowly than the latter, so that the surrounding fundus appears to move over the depressed surface of the disk.

Fig. 323.



Glaucomatous excavation taking place in an optic nerve with a physiological excavation. (Jaeger.)

On examination with the direct method it is found that a different lens is necessary to see clearly the bottom of the cup, from that which is required for the rest of the fundus. Thus supposing the patient to be emmetropic, a minus lens would be needed to see distinctly the bottom of the cup; or if the patient were myopic, a higher minus glass than that used for seeing the rest of the fundus.

When the side of the cup is steep or somewhat overhanging, the

retinal vessels as they curl around it disappear from view for a portion of their course, or may be viewed in a foreshortened manner. If they disappear from view for a portion of their course, there seems to be a break in the continuity of the vessel seen at the base of the cup and on the surface of the retina, it often reappearing at a slightly different position on the latter from what it disappeared at on the former. If viewed in a foreshortened manner, the blood in the vessel appears very dark.

The margin of a cupped disk in glaucoma often throws a shadow on its surface, so that it appears lightest in the centre; the markings of the lamina cribrosa on its surface are usually well defined, and its color is sometimes altered to a greenish or bluish hue.

Conditions which Predispose to Primary Glaucoma or Excite Acute Attacks.

Age. There are cases of glaucoma that date from birth, or even before birth, which are not the result of some obvious precedent disease, and which might be classed as cases of primary glaucoma. They are, probably, the result of some congenital defect in development, and evidently have a different causation to that of other cases of primary glaucoma. They will, therefore, be dealt with separately under the heading of congenital glaucoma or primary buphthalmos. Excluding these cases, it may be stated that the liability to primary glaucoma increases with advance of life. Priestley Smith[1] has shown, from a careful analysis of 1000 cases, that at the age of sixty-five years the chance of an attack of glaucoma is at least one hundred times greater than at fifteen years, and more than twice as great as at forty-five years of age. Primary glaucoma under thirty years is very rare; in practice it is met with most commonly in the decade between sixty and seventy years.

Sex. Women are more liable to primary glaucoma than men, especially to acute attacks.

Heredity. Several striking instances are recorded in which primary glaucoma has occurred in members of the same family, through two or three generations. The number of cases where an hereditary tendency is met with compared with the number of cases of glaucoma which occur are, however, few.

Race. Certain races are stated to be more liable to primary glaucoma than others, viz.: the Jews, the Egyptians, and the negroes of Brazil. More definite statistical evidence is required on these matters before it can be said that they are proved.

Errors of Refraction. Nearly 50 per cent. of eyes affected with primary glaucoma are hypermetropic, and it is generally believed that a hypermetropic eye is predisposed to glaucoma. It should be borne in mind, however, that hypermetropia is the commonest re-

[1] Transactions of the Ophthalmological Society of the United Kingdom, 1880, vol. vi. p. 294.

fractive error, and that the proportion of cases of glaucoma occurring in association with hypermetropia is not very much larger than that of cases of hypermetropia to the population at large.

Glaucoma in cases of high myopia is very uncommon.

Accommodative Effort. There seems good reason to believe that prolonged near work tends to the production of primary glaucoma. Scheon[1] has published statistics to show that the occurrence of glaucoma is often associated with neglect of the use of proper glasses.

Smallness of Cornea. Priestley Smith[2] has proved conclusively that eyes with small corneæ are predisposed to primary glaucoma. He says that eyes in which the cornea measures only ten millimetres in the horizontal diameter seem seldom to escape the disease. He has farther shown that smallness of the cornea means smallness of the eyeball.

Depressing Emotions. Among the more direct causes of primary glaucoma, first and foremost is emotional excitement of a depressing character. Grief connected with the death of a friend or relative, anxiety connected with business matters, or worry and trouble due to other cause, very commonly precedes the onset of glaucoma.

Widows seem especially liable to glaucoma. Thus out of 117 cases 74 were females, 43 males. Of the 74 females, 38 were married, 27 widows, 7 single, 2 unrecorded. Of the 43 males, 31 were married, 8 widowers, 1 single, 3 unrecorded.

Loss of Sleep. Associated with anxiety or trouble, very commonly, is loss of sleep, and the two seem to act together in tending to bring on glaucoma. A by no means uncommon history is that the first symptoms came on after the patient had sat up at night tending some dearly loved sick relative or friend.

Operation on One Eye. All that may be comprised under the term "shock," both mental and physical, which follows an operation for glaucoma on one eye, is very liable to excite an acute attack in the other. The second eye would no doubt be predisposed to glaucoma, but may not previously have evinced any symptoms of the disease.

Mydriatics. The use of a mydriatic such as atropine, to an eye predisposed to primary glaucoma is very liable to cause increase of tension, and its application may excite acute attacks. If used in chronic cases, it may intensify the increased tension and cause acute symptoms to set in.

Local Injuries. A slight injury of the eye, an abrasion or ulcer of the cornea, sometimes seems to be the determining cause of the onset of primary glaucoma.

Constitutional Conditions. An attack of facial neuralgia is sometimes the precursor of glaucoma. Other possible contributing con-

[1] Archiv f. Ophthalmologie, 1887, Band xxxviii., ab. 1, S. 195.

[2] Transactions of the Ophthalmological Society of the United Kingdom, 1890, vol. x. p. 68.

stitutional causes are cold, fatigue, constipation, and vascular or lung affections which produce venous congestion of the head and eyes.

The Clinical Types of Primary Glaucoma.

From what has been said of the effects of increased tension on the different structures of the eye, it will be seen that the symptoms of primary glaucoma vary considerably according to its mode of onset. Different clinical types of the disease may consequently be described: acute congestive, subacute, and chronic or non-congestive.

It must, however, be borne in mind that no hard-and-fast line can be drawn between these different types. They merge into one another. An eye may be affected with one type of the disease at one time and a different one at another; or the same individual may have one type in one eye and a different type in its fellow.

Acute Congestive Glaucoma. An acute attack of glaucoma may occur in a person who has not previously had premonitory symptoms of the disease; it is then spoken of as *fulminating glaucoma.* More frequently it comes on in those who have experienced one or more slight subacute attacks or in a patient who has been suffering from the chronic form of the disease.

The attack is ushered in by sudden aching pain in the eye and forehead, sometimes also of the whole side of the face. This pain is accompanied by a general feeling of malaise, repeated attacks of vomiting, and loss of appetite. So severe sometimes are these general symptoms that they are attributed to "a bilious attack," and their connection with the affection of the eye is often overlooked, even by the medical attendant.

The vision rapidly fails, going on in the course of a few hours or days, according to the severity of the attack, to bare perception of light or complete blindness. The eyelids and conjunctiva become somewhat swollen from œdema. The vessels of the ocular conjunctiva and episcleral tissue, especially the main trunks, become much congested with venous blood. The cornea loses its natural brilliancy, presenting a dull, hazy appearance, and after a short while it is less sensitive to the touch than normal. The anterior chamber is very shallow; the pupil is semidilated and immobile, it commonly assumes a somewhat oval shape, and may be eccentric. The iris viewed through the dull cornea will be less brightly colored than that of the fellow eye. Turbidity of the media usually prevents any view of the details of the fundus being obtained ophthalmoscopically. On palpation of the globe through the lids, the tension will be found considerably raised, usually as much as +2 or +3. If the case is left to itself without treatment, the symptoms of congestion will last some weeks, and then, as the intra-ocular vascular circulation adapts itself to the altered conditions, subside. The tension of the eye, however, remains increased. The subsidence of the congestion is accompanied by relief of pain and some improvement of sight. The cornea

becomes less hazy and a view of the fundus can be obtained ophthalmoscopically, when, if the case has been of sufficient duration, the characteristic cupping of the optic disk will be detected. The anterior ciliary vessels remain enlarged and the pupil dilated, acting very slightly to light. Fresh subacute attacks may follow, after each of which less and less vision is recovered, absolute permanent blindness ultimately resulting.

Sometimes after the first acute attack the glaucomatous condition will become chronic, and the field of vision gradually and steadily contracts without fresh onsets of congestion and pain.

When the eye has become quite blind and the condition of absolute glaucoma is established, the iris will be much shrunken and discolored, the pupil dilated and immobile; at its margin on the surface of the iris there will be a dark ring, often extending farther in one direction than elsewhere—ectropion of the pigment epithelium. The cornea will be dull, and vesicles may form on its surface. It will be very liable to become ulcerated, and the ulcer will be difficult to heal, often going on to perforation, with escape of some of the contents of the globe.

The anterior chamber will continue very shallow, and a dull grayish-green reflex often be seen from the pupil, or the lens may become cataractous. The anterior perforating vessels remain enlarged, and at any weak spots in the walls of the globe the sclerotic will give, becoming staphylomatous and discolored.

Subacute Glaucoma. Subacute attacks of glaucoma, as has been said, may precede or succeed an acute attack. They may also occur independently.

In a subacute attack the symptoms are of a character similar to those in an acute attack, but of less severity. The pain is confined to the eye and is of the nature of a ciliary neuralgia. The patient complains that during the attack there is an appearance as of a fog or mist in front of the sight, and that around lamps at night-time he sees rings of colors like those of a rainbow. The field of vision shows contraction, usually on the nasal side.

The injection of the eye is not very intense; it has a dusky hue, and usually is confined to the anterior perforating and episcleral vessels. There is a slight steaminess of the cornea, especially about its centre, resembling glass which has been breathed upon. The pupil is semidilated and sluggish in action; the anterior chamber is shallow. On ophthalmoscopic examination the optic disk will be found cupped. In an early case the cupping may be very slight, amounting to only a slight abrupt bending of the vessels at one margin.

The tension will generally be found about +1 or +2. A subacute attack may vary in duration from a few hours to a few days.

In some patients the symptoms come on in the evenings, subsiding after a night's rest. In the intervals, at first, perfect vision is restored, or a slight contraction of the field may be the only damage

that has been effected. As the attacks become repeated, more and more permanent damage ensues, the amount depending largely on their severity and duration.

Some cases after one or two subacute attacks settle into a chronic state with a persistent small amount of increase of tension, without further exacerbation of symptoms, except steady contraction of the field of vision.

Chronic Non-congestive Glaucoma. A case of glaucoma may run a chronic course from first to last. A case commencing as chronic glaucoma may become complicated with an acute or subacute attack. A case beginning with an acute or subacute attack may afterward pass into a chronic condition. In chronic glaucoma there is no pain, and the patient is unable to state definitely, in a case commencing as such, when the affection began. The disease may progress to almost complete blindness in one eye in unobservant patients without their knowing that anything is the matter.

An early symptom often is the apparent rapid advance of presbyopia, the patients having to keep on changing and increasing the strength of their glasses for reading.

There is no injection of the eye, or at most a slight increase in the size of the anterior perforating veins. To outward appearances no alteration in the eye is to be observed. The cornea remains bright, and the pupil of normal size and reacting to light. The anterior chamber is usually shallower than normal.

The only subjective symptom is the loss of sight, which commences at the periphery of the field and extends inward. The failure in vision may be exceedingly slow, extending over many years. Often perfect central vision is retained when the field has become contracted close up to the central area in all directions. The degree of tension varies in amount and in different cases.

There are cases in which the tension is hardly ever found to be appreciably increased by the finger-test, but in which the cupping of the optic disk and loss of vision ensue in the same way as in cases of chronic glaucoma, where the increase of tension is undoubted. It is possible that in some of these cases the tension becomes raised only in the evenings, at which times they are not under observation of the surgeon; or it may be that there is abnormal elasticity of the lamina cribrosa, which allows it to be depressed back, with an exceedingly slight increase of the normal amount of intra-ocular pressure.

In other cases of chronic glaucoma the tension varies in degree from time to time; it may be as much as +2.

Ophthalmoscopically, characteristic cupping of the optic disk, with a well-marked scleral ring surrounding the disk, is seen. Pulsation of the retinal arteries in the vicinity of the disk will be present or readily obtained by slight pressure of the finger on the globe. The choroid will present a tessellated appearance.

Diagnosis of Primary Glaucoma.

In cases of acute glaucoma the general disturbance is often so great that the fact that it is all secondary to increased tension of the eye is liable to be overlooked. Cases of acute glaucoma not uncommonly are treated for sick headache, neuralgia, erysipelas, influenza, or toothache, and much valuable time is thereby lost. The rapid failure of vision should serve at once to distinguish glaucoma from such affections. The shallow anterior chamber, dull cornea, semidilated pupil, and increase of tension should determine the diagnosis.

A difficulty sometimes arises in distinguishing cases of subacute primary glaucoma from iritis. The difficulty is much increased when, as occasionally happens, the iritis is associated with increase of tension. In both conditions the iris may be altered in brightness or color. The presence of posterior synechiæ should point at once to iritis, but it may be difficult to decide whether or not any are present without dilating the pupil; and if the case is one of primary glaucoma, it is very desirable not to use a mydriatic. In glaucoma there will not be so much ciliary injection and photophobia as in iritis. The anterior chamber will be shallow, while in iritis it will be of normal depth, or possibly deeper than normal, with dotted opacities on the back of the cornea (*keratitis punctata*). The history of the case may assist in the diagnosis. In primary glaucoma a history of previous attacks of dimness of sight with an appearance of rainbow colors around lights may be elicited; while in iritis there may have been a recent attack of syphilis or gonorrhœal arthritis, or previous attacks of similar inflammation in the eye itself or the fellow eye. Differentiation between these two affections is of importance in deciding whether to prescribe a myotic or a mydriatic.

Confusion sometimes occurs in distinguishing between cupping of the optic disk due to glaucoma, cupping due to atrophy, and cupping the result of a physiological peculiarity.

As already stated, the glaucomatous cup is due to atrophy of the nerve fibres down to the lamina cribrosa and depression backward of the lamina cribrosa. In an atrophic cup there is no depression backward of the lamina cribrosa, only atrophy of the nerve fibres down to it.

Both the glaucomatous cup and the atrophic cup occupy the whole area of the optic disk. The latter, however, has gradually shelving edges, while the former has steep sides or overhanging edges, around which the retinal vessels disappear for a portion of their extent.

The physiological cup is produced by divergence of the nerve fibres as they enter the eye after piercing the lamina cribrosa. This divergence occurs sooner or is more marked in some eyes than in others; then a cup or depression is found in the head of the nerve. It is

distinguished from the other two forms of cupping by never occupying the whole area of the optic disk. Like a glaucomatous cup, it sometimes has an overhanging edge, around which the retinal vessels are seen to curl; this may at times lead inexperienced observers into making an erroneous diagnosis, which can be avoided by observing that only a portion of the disk is involved.

The diagnosis between cases of chronic glaucoma, where the tension is not increased at the time the patient is seen, and cases of primary optic atrophy, is not always easy. Besides the differences in the character of the cupping of the nerve just mentioned, pulsation of the retinal arteries in the vicinity of the disk should be looked for. Its presence should point at once to glaucoma. If absent, a slight amount of pressure should be made on the globe with the finger, to see if it can easily be evoked.

The character of the field of vision may be of considerable assistance in distinguishing between these two affections. A contraction of the inner part of the field is a characteristic symptom of glaucoma. The field in both conditions may be concentrically contracted or present sector-shaped defects. In atrophy the fields for color will be contracted out of proportion to that for white, or there may be complete color blindness; while in glaucoma the contraction of the fields for color is always concentric with that for white, and color blindness is met with only in the latest stages of the disease.

Varieties of Secondary Glaucoma.

As the result of various different diseased conditions of the eye, a derangement of the circulation of the intra-ocular fluid is liable to occur, resulting in increased tension.

The different diseases in connection with which secondary glaucoma occurs are dealt with in detail in other portions of this work; it is necessary here only to enumerate them, and to point out the manner in which each interferes with the circulation so as to produce the glaucomatous condition.

Posterior Synechiæ of Iris. As the result of iritis, the pupillary margin of the iris may become adherent to the lens capsule in its entire circumference, a condition spoken of as annular posterior synechia. The aqueous humor then is obstructed in its passage forward through the pupil into the anterior chamber. It accumulates between the iris and lens, bowing the former forward and producing the condition termed iris bombé. At first this accumulation of fluid behind the iris is compensated for by escape of fluid from the anterior chamber. Ultimately the root of the iris comes into contact with the back of the cornea, so that not only is passage of fluid through the pupil obstructed, but also any escape through the spaces of Fontana. The tension then becomes increased.

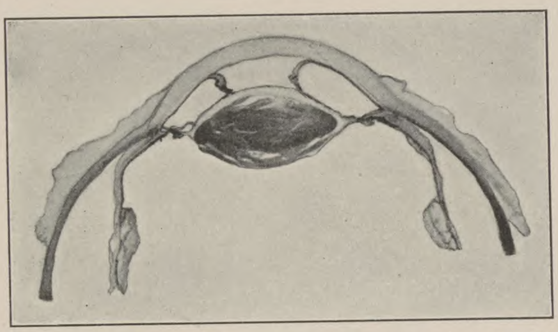
This is a form of secondary glaucoma which is readily relieved

by iridectomy. When a portion of the iris is removed, fluid can again pass forward into the anterior chamber and the normal circulation will be re-established.

Sometimes as the result of iritis, not only the pupillary margin, but also the whole of the posterior surface of the iris becomes united to the lens capsule, a condition known as *total posterior synechiæ.* Where this is present, the secretion of the ciliary body is unable to find its way forward between the iris and lens. It accumulates in the vitreous chamber, increasing the pressure there, and forcing forward the lens and iris. When the root of the latter comes into apposition with the periphery of the cornea, farther escape of fluid from the anterior chamber through the filtration area is blocked, and glaucoma becomes established.

The relief of tension in this condition by iridectomy is not an easy matter. It is very difficult to remove a piece of iris which will allow

FIG. 324.



Glaucoma secondary to iritis and the formation of annular posterior synechia. Fluid accumulating in the posterior chamber has bowed the iris forward into contact with the back of the cornea.

of the satisfactory passage of fluids forward, so firmly is it bound down and so frail does its tissue become.

Anterior Synechiæ of Iris. The formation of an extensive adhesion of the iris to the scar tissue left after a perforating ulcer of the cornea may prevent passage of fluid through the pupil, so that the whole anterior chamber becomes obliterated. The aqueous humor accumulates between the back of the iris and lens; none can gain exit from the eye through the spaces of Fontana or through the lymph spaces on the anterior surface of the iris. The tension then is increased, as the result of which the recently inflamed and softened corneal tissue gives and becomes staphylomatous.

A less extensive adhesion of the iris to the cornea, which does not involve the entire circumference of the pupil, may cause it to be so drawn forward as to bring its root into contact with the back of the cornea. By an obstruction to the passage of fluid through its main exit at the angle of the anterior chamber, glaucoma is established.

Such an obstruction is especially liable to occur when a cornea becomes staphylomatous, as an increase in the prominence of it tends to draw the iris still farther forward.

Anterior Synechiæ of Lens Capsule or Hyaloid Membrane of Vitreous. After the operations of extraction of cataract, or discission, or after wounds of the eye, adhesions of the capsule or portions of the vitreous humor to the cornea are liable to form. These structures, normally situated behind the level of the iris, when advanced in position in this way, may so draw it forward as to bring its root into contact with the back of the cornea, blocking the filtration area.[1] Glaucoma may thus be produced, even when iridectomy has been performed, the filtration area opposite the coloboma being blocked by a small piece of the root of the iris, which has been left, or by the most anterior of the ciliary processes. Glaucoma has occurred

FIG. 325.

Glaucoma secondary to the formation of an anterior synechia of the lens capsule after extraction of cataract. An iridectomy had been performed, but in the region of the coloboma the filtration area is blocked by the anterior of the ciliary processes, drawn forward by the adherent capsule. On the opposite side the angle of the anterior chamber is blocked by a broad adhesion of the root of the iris.

when the whole of the iris has escaped through a wound (traumatic aniridia[2]), the filtration area being blocked in its entire circumference by the anterior of the ciliary processes, drawn forward by reason of adhesion of the lens capsule to the cornea.

Wounds of the Lens. Increase of tension sometimes results from swelling of the lens substance in its capsule, after some of the aqueous humor has been admitted to it through a wound. In such cases the swollen lens seems directly to press forward the iris and close the filtration area. The tension can usually be relieved by making a freer opening in the capsule and allowing some of the lens matter to escape from the eye or into the anterior chamber.

1 Transactions of the Ophthalmological Society of the United Kingdom, 1890, vol. x. p. 108.
2 Ophthalmic Review, 1891, vol. x. p. 105.

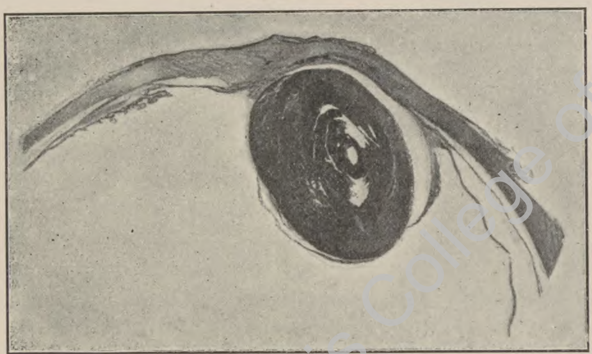
Increase of tension may also occur after wounds of the lens, when there has been a free opening made in its capsule and some of the lens matter has come forward and dissolved in the aqueous humor.

FIG. 326.

Glaucoma secondary to dislocation of the lens into the anterior chamber. The lens was in contact with the cornea, and the iris closely pressed forward into contact with the periphery of the cornea and back of the lens. In the preparation of the specimen the structures have fallen slightly apart. The abrupt bend in the iris, where it ceased to be in contact with the cornea and passed into contact with the lens, is well shown.

After the operation of discission for lamellar cataract increased tension is of not uncommon occurrence. The aqueous humor becomes loaded with the colloid substance globulin, of which the lens is mainly

FIG. 327.



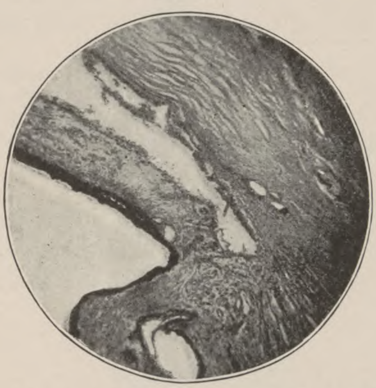
Glaucoma secondary to traumatic dislocation of the lens. The displaced lens has pressed forward the iris into contact with the back of the cornea. The iris and ciliary body where they were in apposition with the lens are much atrophied. In the preparation of the specimen an obvious slight alteration in the position of the lens has taken place.

composed; it is then much less easy for it to filter out of the eye than in its normal condition. It is possible also that bits of undissolved lens substance may become entangled in the mesh of the ligamentum pectinatum and fill up the spaces contained in it. Increased tension brought about in this way is always readily relieved by a paracentesis and evacuation of the lens matter.

Dislocation of the Lens. Glaucoma is a not infrequent accompaniment of dislocation of the lens. Cases are met with in which the suspensory ligament is deficient in a portion of its circumference, and the remainder so attached as to allow the lens to sway backward and forward with movements of the head. In some of these, when the patient hangs his head down and the lens falls forward, the ocular tension becomes increased, returning, however, to normal when he raises his head and the lens falls back.

Similarly, permanent displacement forward of the lens into the anterior chamber, either completely or partially, causes increase of tension. The displaced lens fills up the pupil and blocks the passage of fluid through it. The aqueous humor then accumulates in the vitreous chamber. (Fig. 326.)

FIG. 328.



Angle of the anterior chamber in an eye which had glaucoma secondary to serous iridocyclitis. It shows wide separation of the root of the iris from the back of the cornea, but an accumulation of inflammatory cells on the inner surface of Descemet's membrane and in the mesh of the ligamentum pectinatum.

When the lens is displaced backward or laterally and increased tension results, the angle of the anterior chamber is always found closed. The relative alteration in position of the lens and vitreous body seems to force directly forward the root of the iris. (Fig. 327.)

Serous Cyclitis. When the ciliary body is inflamed, the fluid secreted by it is in excess and altered in character; it is more serous than normal, and is mixed with a number of formed elements, leucocytes, and cells which have desquamated from the secreting surface. The increased amount of albumin in the fluid makes it more difficult for it to filter out of the eye. The formed elements in it, as they pass through the ligamentum pectinatum, get caught in the mesh, and, accumulating there, cause obstruction. (Fig. 328.)

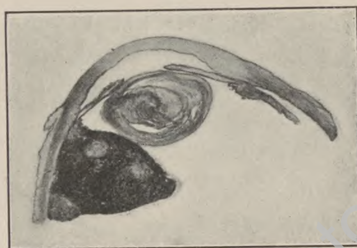
There are then these three factors which combine to give rise to increased tension in serous cyclitis: (1) excess of secretion; (2) albuminous character of the aqueous humor; (3) accumulation of inflammatory cells in the spaces of Fontana.

The obstruction to the circulation of the fluid being primarily at the outlets from the anterior chamber, and the albuminous character of the fluid making it difficult for it to filter through the anterior hyaloid membrane into the vitreous, an accumulation takes place in the anterior chamber, which becomes deepened, the lens and iris being depressed backward. The glaucoma in such cases is treated best by repeated paracentesis, rather than iridectomy,

Intra-ocular Tumors. A tumor growing forward from the retina, or beneath the retina from the choroid, tends to increase the pressure in the vitreous chamber. This may for a time be compensated for by an increased escape of fluid from the vitreous into the anterior chamber, and an increased escape from the eye. Gradually the vitreous becomes so compressed that fluid is less and less easily pressed out of it. The tension then in the vitreous chamber becomes greater than in the anterior, the lens and iris are pushed forward until the root of the latter comes in contact with the filtration area in the cornea, and a permanent block to the escape of fluid from the eye is established. (Fig. 329.)

In a case of sarcoma of the iris or ciliary body, which has caused increase of tension, it will generally be found that the spaces of Fontana contain cells similar in character to those of the growth. They are, naturally, most numerous in the part in the vicinity of the growth, but may also be met with in parts quite remote from it. The angle of the anterior chamber may also be found closed, by the root of the iris having been directly pushed forward by the growth, or by thickening of the root of the iris by the growth itself.

FIG. 329.



Glaucoma secondary to melanotic sarcoma of the ciliary body. The lens is shown directly pressed forward by the growth. The angle of the anterior chamber is closed by contact of the root of the iris and cornea.

Epithelial Cysts in the Anterior Chamber.[1] When an opening has been made into the anterior chamber, either by an operation or accidentally, some of the surface epithelium may be implanted or spread along the wound into the anterior chamber. The epithelium, subsequently forming a cyst which fills the whole anterior chamber, obstructs the passage of aqueous humor into it, and causes increase of tension.

Detachment of the Retina. Glaucoma sometimes comes on in eyes with simple detachment of the retina. When this is the case, the presence of an intra-ocular growth is generally suspected, and often it is not until the removal of the eye that it is ascertained definitely that the condition is one of simple detachment. The extent of the detachment is usually great, and the vitreous much shrunken. Some hemorrhage or serous effusion into the subretinal space forces forward the lens and iris, blocking the angle of the anterior chamber, much in the same way as in the case of an intra-ocular growth.

Thrombosis of the Retinal Veins. The condition which is commonly spoken of as "hemorrhagic retinitis" is probably in most cases due to thrombosis of the central retinal vein; it may sometimes

[1] Transactions of the Ophthalmological Society of the United Kingdom, 1892, vol. xii. p. 175.

be the precursor of an attack of glaucoma. The symptoms of the attack resemble those of a case of primary glaucoma, from which it can only be distinguished by the presence of numerous hemorrhages scattered over the retina, and the enlargement of the retinal veins.

Thrombosis of the retinal vein is mostly met with in elderly people, and may exist without any increase of ocular tension.

It seems probable that it is only in those eyes which are structurally predisposed that glaucoma is set up, increase of pressure in the vitreous chamber being the exciting cause. The venous engorgement and serous effusion into the retina, together with the serous effusion into the vitreous which follows thrombosis, cause this increase of pressure. In such cases the iris and lens are pressed forward and the angle of the anterior chamber closed. It is a condition which it is very necessary to differentiate from primary glaucoma, because operative procedures on such eyes are followed frequently by extensive and disastrous hemorrhage.

Where possible, operations in such cases should be avoided, preference being given to treatment with myotics. If an operation becomes urgently necessary, a paracentesis or sclerotomy should be performed, the escape of fluid and lowering of tension being effected as gradually as possible.

Congenital Aniridia and Coloboma of the Iris. That glaucoma can supervene when there is apparently entire congenital absence,

FIG. 330.

Congenital aniridia in which glaucoma followed on a perforating ulcer of the cornea. The angle of the anterior chamber is shown blocked by a very rudimentary iris, which was so small as not to be visible clinically. It has become adherent to the back of the cornea.

or a coloboma of the iris, seems at first wholly out of keeping with the fact that an iridectomy is the most effective treatment for the relief of tension. Several cases of glaucoma, both primary and secondary, in association with these defects have been recorded.[1] (Fig. 330.) Pathological examination of eyes with these defects has

[1] Ophthalmic Review, 1891, p. 101; Transactions of the Ophthalmological Society of the United Kingdom, 1893, vol. xiii. p. 128.

shown that the ciliary body really ends in a small rudimentary iris, which, though not of sufficient length to render it visible beyond the sclerocorneal margin, is of sufficient size when pressed forward to block the filtration area. In two cases where increased tension was present the rudimentary iris was actually found blocking the filtration area.

There is reason to believe that cases with congenital defects of the iris may be predisposed to glaucoma. Strands of tissue have been found stretching from the anterior surface of the defective iris to the ligamentum pectinatum, showing a congenitally imperfect separation of these structures.

Congenital Glaucoma, or Primary Buphthalmos.

Enlargement of the cornea, and the production of a condition resembling a bullock's eye, may occur as the result of increased intraocular tension in children, in whatever way that increase is brought about.

There is a form of buphthalmos that is not the result of any obvious precedent disease, and that may be termed primary. In many such cases a definite history of the symptoms dating from birth can be obtained, and probably so in all, the symptoms in some at first being so slight as to escape observation.

The increase of tension is unaccompanied by injection or other acute symptoms. The enlargement of the globe continues steadily without pain. It is not only the cornea that is increased in size, but the whole eyeball in all its meridians as well.

The measurements of the eye of a boy, aged four years, which was affected in this way, were: antero-posteriorly 28 mm., vertically 26.5 mm., while the diameter of the cornea was 14.5 mm. Besides having its diameters increased, the cornea becomes more convex and globular in shape.

The stretching of the cornea and sclerotic occasions thinning in the latter. This thinning allows of the pigment of the uveal tract being seen through, so that it appears of a bluish-gray color.

The anterior chamber becomes very deep, and the iris is often tremulous on movements of the eye, from weakening of the suspensory ligament or fluidity of the vitreous behind it. Ophthalmoscopically, the optic disk is found deeply cupped.

In some cases the increased tension persists, and failure of sight steadily progresses until the eye becomes quite blind. In others, a spontaneous relief of tension occurs, and, although the eye remains permanently enlarged, there is no further increase in size or deterioration of sight.

A congenital malformation[1] in the channels of exit of fluid from

[1] Treacher Collins, Researches into the Anatomy and Pathology of the Eye, p. 104. F. R. Cross, Transactions of the Ophthalmological Society of the United Kingdom, vol. xvi. p. 304.

the eye is the probable cause of the increase of tension in these cases. In some a congenital adhesion, or, rather, failure in separation of the periphery of the iris and back of the cornea, has been found, in some, strands of tissue about the angle of the anterior chamber, suggestive of adhesions which had become stretched and broken down by the collection of fluid in the anterior chamber, which forced backward the iris and forward the cornea. Such a breaking down of congenital adhesions which at one time existed, explains the cases in which spontaneous relief of tension occurs. In other cases the canal of Schlemm was stated to be congenitally absent.

The operations of iridectomy and sclerotomy in these cases frequently fail to relieve tension or to check the expansion of the globe,

FIG. 331.

The angle of the anterior chamber in a case of congenital glaucoma or primary buphthalmos. Showing a congenital adhesion of the root of the iris to the back of the cornea. The rest of the iris is widely separated from the cornea, the anterior chamber having been very deep.

and are attended with greater risk than in cases of glaucoma in the adult.

The expansion of the globe causes stretching and weakening of the suspensory ligament of the lens, which renders it very liable to rupture on the escape of aqueous from the deepened anterior chamber. Dislocation of the lens or loss of vitreous, which is usually of a fluid consistency, are complications, therefore, likely to occur.

Repeated paracentesis and the use of myotics have been employed as alternative measures, and, although attended with less danger, are frequently equally unsuccessful in checking the progress of the affection.

The appearances of some of the eyes, which have been removed for this condition, suggest that the adherent tags of tissue about the root

of the iris might be disengaged or incised by the point of a knife passed into the extreme angle of the chamber. Such an operation has been practised by Vincenti[1] for glaucoma in the eyes of older people.

Pathogenesis of Primary Glaucoma.

The whole sequence of events which result in the production of increased tension in primary glaucoma is by no means so obvious as in some of the secondary forms of the affection. It is needless here to enter into the numerous theories which have been suggested as to the origin of this disease, many of which, with the growth of knowledge respecting the intra-ocular circulation, have been shown to be untenable.

Any increase of secretion into the eye is compensated for by an increased outflow, so that no theory of the pathogenesis of glaucoma based on an increased secretion alone can be accepted. We must look to the channels of exit of fluid from the eye for some obstruction to account for the increased intra-ocular pressure.

As pointed out by Max Knies and Weber, the filtration area at the angle of the anterior chamber in primary glaucoma is found obstructed by apposition or adhesion of the root of the iris to the periphery of the cornea, the iris apparently being pushed forward by pressure of the ciliary processes against its root.

If a block to the exit of fluids from the eye at the angle of the anterior chamber is the primary cause of glaucoma, we should expect to meet with an accumulation of aqueous humor and a deepening of the anterior chamber. Instead of this, we find the anterior chamber shallowed, the shallowness often preceding the onset of increased tension.

Priestley Smith has met this difficulty by suggesting that there is at first an accumulation of fluid in the vitreous chamber, due to obstruction at the circumlental space, which causes the lens, iris, and ciliary processes to be pressed forward. He has shown that changes which predispose to narrowing of the circumlental space predispose to glaucoma. It will be well here to quote his words in which he sums up his views on this matter:[2]

"Primary glaucoma appears usually to depend on some vascular disturbance which congests the uveal tract, or upon a faulty relation of the lens to the parts around it, or upon both. If the patient be elderly, we know that the lens is relatively large. If the cornea be small, we may infer that the whole eyeball is small, and that the relations of the lens are such as to predispose to compression of the filtration angle, especially during dilatation of the pupil. An obstruction in the region of the hyaloid and the circumlental space, which checks the escape of surplus fluid from the vitreous and leads to an

[1] Revue générale d'Oph., November 30, 1894.

[2] Norris and Oliver, System of Diseases of the Eye, vol. iii. p. 656.

advance of the lens, appears to be present in many cases. Slackness of the zonular, with consequent instability of the lens, is probably a contributory cause. Through one or other of these causes, or several in combination, the ciliary processes are pressed against the iris, and the filtration angle is narrowed or closed."

Panas, Jonnesco, and others look for an explanation of the increased tension in glaucoma to some disturbance in the controlling action of the nervous system on the intra-ocular pressure. As already stated, our knowledge of this controlling action is at present incomplete.

The reduction of tension in glaucoma which Jonnesco has been able to effect by resection of the superior cervical sympathetic ganglion has led him to formulate the theory that glaucoma is due to peripheral or central irritation, either permanent or intermittent, of the ocular sympathetic fibres which pass through it. The effects on the eye of such irritation have been mentioned. The dilatation of the pupil, which is thereby produced, might alone, in an eye with a shallow anterior chamber and predisposed to glaucoma, be sufficient to bring on increase of tension, just as atropine mydriasis sometimes does.

The influence which emotional disturbances sometimes have in the production of glaucoma may possibly in this way find an explanation.

Treatment of Primary Glaucoma.

Fifty years ago glaucoma was an incurable disease. Until von Graefe introduced the treatment of it by iridectomy in 1856, no means which would afford permanent relief was known.

In 1876 the next most valuable method for the reduction of increased tension, viz.: the use of myotics, was suggested by Laqueur, of Strasburg.

These two chief forms of treatment, together with other procedures for the reduction of tension, will now be individually described, and afterward their clinical application.

Iridectomy or Iridectomydialysis. The way in which an iridectomy relieves tension in primary glaucoma has been the source of much discussion. The pathological examination and comparison of eyes in which it has proved successful, with those in which it has failed, have thrown much light on this matter.[1]

Several eyes have been examined in which an iridectomy successfully relieved the tension in glaucoma and subsequently had to be removed for some intercurrent malady. In these eyes either the obstructed passage for the exit of fluid at the angle of the anterior chamber was found opened up, or a new channel of exit had been established by the formation of what is termed a cystoid cicatrix. (Fig. 332.)

The opening up of the filtration area at the angle of the anterior chamber had in some of the cases been effected by removal of the

[1] Royal London Ophthalmic Hospital Reports, 1891, vol. xiii. p. 166.

obstructing iris up to its point of junction with the ciliary body, in the vicinity of the wound. In others, although a portion of the root

FIG. 332.

Section through the centre of the coloboma in an eye which had had an iridectomy performed for glaucoma of two months' standing. The tension was relieved by the operation. The eye was excised five weeks later for ulceration of the cornea. The iris has been removed up to the ciliary body and the angle of the anterior chamber thereby opened up.

FIG. 333.

Section through a cystoid cicatrix, which formed at the angle of a coloboma in an eye in which glaucoma came on after extraction of cataract. It shows a fistula in the sclerocorneal tissue lined by a fold of atrophied iris. The subconjunctival tissue around the fistula is swollen.

had been left, it became dislodged from its faulty position. Evidently in such cases sufficient time had not elapsed for it to become

adherent to the cornea. The drag on the iris, escape of aqueous, and consequent relief of pressure in the vitreous chamber, together with the local escape of blood, had sufficed to restore the normal channels for the circulation of fluid. (Fig. 333.)

When a cystoid cicatrix is present, a fistula is established in the fibrous tissue at the sclerocorneal margin, through which fluid may pass from the anterior chamber into the subconjunctival tissue and be absorbed there by the conjunctival vessels. The tissue around the fistula is found usually in a boggy condition.

The fistula results from prolapse of a fold of iris, which prevents the two sides of the wound in the fibrous tissue of the sclera and cornea from uniting, but over which the conjunctiva heals. At first

Fig. 334.

Section through a cystoid cicatrix which formed after an iridectomy for chronic glaucoma. The tension remained normal for a year; the eye was then excised on account of iritis and pain. A fistula in the sclerocorneal tissue is shown, lined partly by the atrophied root of the iris, and partly by the ciliary processes. The subconjunctival tissue around the fistula is swollen.

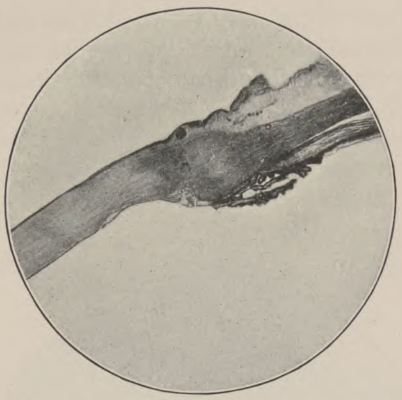
the iris tissue lining such tract offers an impediment to the passage of fluid out of the eye; but being a weak spot in the globe, it tends to bulge, and the iris lining it atrophies, until ultimately a fistula is established. (Fig. 334.)

A cystoid condition of a cicatrix after iridectomy appears most often at the angle of the coloboma—*i. e.*, the position where the prolapse of a fold of iris is most likely to occur.

Although the production of such a condition may prove beneficial in relieving tension, it is one which is attended with a certain amount of risk. What is practically an adhesion between the conjunctiva and iris being formed, any inflammation of the former readily spreads to the latter, and is liable to start a general uveitis.

An iridectomy fails to relieve tension in primary glaucoma when the normal passages for the exit of fluid from the eye remain unopened up, and no new channel is formed.

Fig. 335.



Section through the centre of the coloboma in an eye with absolute glaucoma which had had an iridectomy performed, and in which the increased tension subsequently returned. It shows the cicatrix to be a very peripheral one and free from any entanglement of the iris. The angle of the anterior chamber remains blocked by a portion of the root of the iris, intimately adherent to the cornea.

Fig. 336.

Section through the centre of the coloboma in an eye which had had an iridectomy performed for subacute glaucoma, and in which the increased tension had subsequently returned. It shows that a large portion of the periphery of the iris had been left blocking the infiltration area. The cut end of the iris is adherent to the corneal cicatrix. It looks as though the root of the iris might have been torn away much nearer up to the ciliary body. Probably a simple iridectomy was performed, not an iridectomydialysis.

The normal passages for the exit of fluid at the angle of the anterior chamber are unopened up:

1. When the root of the iris has become so intimately adherent to the back of the cornea that on being drawn upon, instead of tearing at its extreme root, it tears through at the point where it ceases to be adherent, and the portion causing obstruction is left behind. (Fig. 336.)

2. When by reason of the way in which the iridectomy has been performed a portion of its root is left behind, which, though not adherent to the cornea, has failed to become dislodged from its faulty position. (Fig. 337.)

3. When by reason of delayed reformation of the anterior chamber the lens becomes united to the posterior surface of the wound by plastic exudation thrown out from the latter. Then, when the

Fig. 337.

Section through the centre of the coloboma in an eye which had had an iridectomy performed for absolute glaucoma. An adhesion of the lens capsule to the posterior surface of the cornea in the region of the cicatrix is shown. The lens has become somewhat displaced backward in the preparation of the specimen. The angle of the anterior chamber is closed by the anterior of the ciliary processes, which have been pressed forward by the adherent lens.

anterior chamber does reform, the lens becomes drawn forward, its adherent margin pressing the anterior of the ciliary processes into contact with the filtration area in the region of the coloboma.

In the absence of prolapse of a fold of iris, no fistula and no new channel of exit for fluid are produced. From the foregoing, it is obviously desirable in performing an iridectomy for glaucoma to try to remove the extreme periphery of the iris up to its point of junction with the ciliary body. Fortunately it is at this spot that the iris is thinnest, and it is here that it is most likely to tear through when drawn upon, unless abnormally adherent.

In performing an iridectomy for glaucoma, after an incision of the sclerocorneal margin has been made, the iris should be drawn out and snipped through from its pupillary to its ciliary margin at one

angle of the wound. It should then be drawn along the whole length of the wound, so as to tear it away, and, finally, cut through again at its further extremity. This method of performing an iridectomy is very different from that adopted preliminary to the removal of a cataract or to form an artificial pupil. For these purposes a piece of iris is simply drawn out of the wound and snipped off.

To distinguish the two methods of performing the operation, the one from the other, it is well to term the first an iridectomydialysis, and the latter simple iridectomy.

Anterior Sclerotomy. Anterior sclerotomy of de Wecker is performed by entering a Graefe knife 1 mm. external to the corneal margin, carrying it across the anterior chamber, and bringing it out equidistant on the opposite side. The points of entrance and exit are planned as if a flap $2\frac{1}{2}$ mm. high were about to be cut. After the

FIG. 338.

Section through the angle of the anterior chamber of an eye with absolute glaucoma, upon which an experimental anterior sclerotomy was performed, 2 mm. distant from the sclerocorneal margin, immediately after enucleation. It shows that the periphery of the iris had been divided in two places, the knife having passed through the adherent root of iris into the posterior chamber.

knife is inserted, it is drawn backward and forward with a sawing motion, but is taken out before a flap has been completely cut. A bridge of tissue is left between the two cuts, composed of conjunctiva and part of the sclerotic. Prolapse of iris very frequently follows the operation, and it is recommended that any tendency to it should be counteracted by the use of eserine before and after the operation.

The operation is designed to form a cicatrix at the sclerocorneal margin, without the removal of any iris. Its value in producing a permanent relief of tension in primary glaucoma has proved to be far inferior to iridectomy, and the results obtained by it are very uncertain.

Experimentally and pathologically it has been shown that a mere band of cicatricial tissue at the sclerocorneal margin does not allow of filtration of fluid through it.

When prolapse of a fold of iris follows a sclerotomy, a fistula and cystoid condition of the cicatrix may be formed, as after iridectomy. In this way an artificial channel for exit of fluid will be formed and the tension relieved.

In an eye which had been enucleated for absolute glaucoma this operation was performed immediately afterward. Sections subsequently made through the seat of operation showed that at the points of puncture and counter-puncture the root of the iris had been divided in two places. (Fig. 338.) The knife had passed from the sclerotic through the iris just where it ceased to be adherent to the cornea into the posterior chamber, and then through the iris again into the anterior chamber. By cutting across the adherent root of the iris in this way the operation may possibly open up a passage for fluid from the posterior chamber into the spaces of Fontana.

An operation termed iridosclerotomy or scleroiritomy, in which the adherent root of the iris is intentionally cut through, has been practised by Knies and Nicati.

Scleral Puncture of Posterior Sclerotomy. Posterior sclerotomy is not an operation from which alone permanent relief of tension

FIG. 339.

Section of an eye upon which a posterior sclerotomy was performed for glaucoma, and which subsequently became much inflamed. The gap in the coats of the eye is shown, with a tag of vitreous humor, much infiltrated with round cells prolapsing through it. The prolapsed vitreous evidently formed the track along which infection gained entrance to the eye.

may be expected. It may, however, in certain cases, be advantageously employed to produce a temporary effect, or as a preliminary procedure to iridectomy.

The puncture is made with a Graefe knife, which is inserted in such a way as to make an opening meridianal to the corneal margin; for in such a wound there is less tendency to gape than in one made

parallel to its margin, and the choroidal vessels and nerves, which run for the most part antero-posteriorly, are less likely to be divided. A spot is chosen for the operation on the surface of the globe, behind the ciliary body and between the recti muscles, usually up and out, 6 mm. or more posterior to the corneal margin. Before inserting the knife the conjunctiva is drawn with fixation forceps a little to one side, so that on conclusion of the operation the opening in it and in the walls of the globe shall not coincide in position. In withdrawing the knife it is given a half-turn, which makes the wound gape and allows of escape of the fluid.

The hemorrhage which results, both intra-ocular and extra-ocular, is usually insignificant in amount. A prolapsed portion of the vitreous humor (Fig. 339) in the wound has been known to form the track along which septic infection gained entrance into the eye. The escape of fluid from the vitreous, the immediate result of the operation, allows of diminution of pressure in the vitreous chamber and the recession of the lens and iris, with increase in the depth of the anterior chamber. For a short while after the operation fluid may continue to ooze from the wound. Soon, however, it commences to heal, further drainage is arrested, and the tension again becomes increased.

In posterior sclerotomies performed experimentally on rabbits, the wound was found to be closed and any outflow of fluid checked on the eighth day (Tobler[1]).

Paracentesis. Paracentesis of the anterior chamber is another operative measure which affords a temporary relief of tension, and which may sometimes be employed in cases of emergency. As soon as sufficient time has elapsed for a fresh secretion of the intra-ocular fluid to accumulate, the tension becomes re-established. The amount of relief is greatest, and lasts longest, when the anterior chamber is deep and a large amount of fluid can be evacuated, as in cases of glaucoma secondary to serous iridocyclitis.

Removal of the Superior Cervical Ganglion of the Sympathetic. Removal of the superior cervical ganglion of the sympathetic has been shown by Jonnesco, of Bucharest, to be an operation capable of reducing the tension in glaucoma. The results of the operation in eight cases he summarized as follows:

1. Immediate and lasting reduction of tension.
2. Marked and permanent contraction of the pupil, even in the cases in which iridectomy had been previously performed.
3. Absence of frontal headache.
4. Disappearance of the attacks of irritative glaucoma.
5. Considerable permanent improvement in vision in all cases in which complete atrophy of the nerve had not set in.

In other surgeons' hands, though the operation has sometimes been attended with success, disastrous results have also occurred. Some patients have died as the result of the operation. In others

[1] Archives of Ophthalmology, March, 1901.

there have been marked flushing of the side of the head and face and severe pain after the operation. The increased tension, although for a time reduced, has returned in some cases. Symptoms of tachycardia and exophthalmos have also developed.[1]

The ganglion can be reached either by an incision made along the anterior or the posterior border of the sternomastoid muscle. The former is the simpler and causes less injury. Jonnesco recommends that the carotid sheath be opened, the vein separated from the artery, and the ganglion found behind it. Burghard says he has found it much simpler to expose the outer edge of the carotid sheath, and then, with a blunt hook, to pull the sheath and its contents inward toward the median line, when the ganglion is at once exposed. It is then drawn forward with forceps and cut out with scissors.

Myotics. Neither myotics nor mydriatics dropped into a normal eye produce any alteration in the tension which is appreciable by the finger test. Atropine dropped into an eye with a shallow chamber and predisposed to glaucoma, as already stated, may bring on increased tension. Eserine in many cases of primary glaucoma will reduce the tension to normal.

These drugs influence tension appreciably only when the anterior chamber is shallow, where an increased or diminished thickness of the iris is capable of causing apposition, or withdrawal of apposition, of its root with the back of the cornea.

In acute cases of glaucoma, where the sphincter muscle of the iris, from pressure on the ciliary nerves, is paralyzed, myotics fail to cause contraction, and, consequently, are unable to relieve tension. In glaucoma of long standing, where the root of the iris has become absolutely adherent to the back of the cornea and does not simply lie in apposition with it, myotics are unable to separate the adhesion, and in these cases fail also to reduce the abnormal tension.

Eserine, besides contracting the pupil, tends, especially in some people, to cause a certain amount of hyperæmia and irritation or pain. It should, therefore, not be used stronger or more often than is absolutely required to produce the desired effect on the pupil and tension. Solutions of 0.125 per cent. or 0.25 per cent. of the sulphate are most frequently employed. One or two applications of a 1 per cent. solution will sometimes, however, reduce tension when the weaker ones have failed.

When the use of eserine has to be persisted in for some time, it is well to combine with it cocaine. Cocaine has the opposite effect to eserine: it dilates the pupil, diminishes the sensibility of the eye, and contracts the bloodvessels. If a solution be employed containing 0.25 per cent. of sulphate of eserine and 1 per cent. of hydrochloride of cocaine, the myotic effect of the eserine will predominate, but its irritating and hyperæmic effects will be reduced.

Pilocarpine is a feebler myotic than eserine, but causes less irritation.

[1] Herbst, Thèse de Paris, 1900.

It may be used in the form of the nitrate in solutions of 0.5 or 0.75 per cent.

Morphine administered hypodermically, by reason of the myotic effect it produces and its sedative action, often proves a valuable additional aid to other measures in the reduction of increased tension.

Clinical Application of Treatment in Glaucoma. In cases of acute glaucoma iridectomy should be performed at the earliest possible moment; a few hours' delay may make considerable difference in the amount of sight which will be regained.

Eserine should be dropped into the eye two or three times while the patient is being prepared for operation, so as to obtain as much contraction of the pupil as possible. It greatly facilitates the grasping of the iris with forceps and the withdrawal of a portion from the eye. The congestion of the eye and the increased tension prevent cocaine producing any marked anæsthetic effect, so that a general anæsthetic has to be administered. Chloroform is much to be preferred, at any rate while the operation is being performed, as with it there are less venous congestion and less heaving respiratory movements than with ether.

In the performance of any intra-ocular operation, either for acute or chronic glaucoma, an endeavor should be made to lower the increased tension as gradually as possible, so as to avoid any sudden rush of blood into the intra-ocular bloodvessels, causing their rupture and hemorrhage. In making an incision into the anterior chamber, the aqueous should be allowed to drain away, and not to escape with a gush.

After an iridectomy a compress should be applied and a bandage firmly adjusted. The patient should be put to bed and kept there until the anterior chamber has well reformed. Care should be taken to prevent the patient rubbing or touching the eye when half asleep, by tethering the hand on the side operated on to the foot of the bed, so that it cannot be moved beyond a certain safe distance from the eye. To prevent the shock of the operation exciting an acute attack of glaucoma in the fellow eye, eserine drops should be applied to the latter immediately after the operation, and twice a day for the succeeding week.

The prognosis in acute glaucoma, if the operation is performed sufficiently early, is good. If it has been delayed for some days, although the operation may relieve tension, the lost vision will not be restored. In the most acute cases vision may be reduced to no perception of light for a few hours, and then restored to almost its normal acuity. If, however, there has been no perception of light for two or three days, the chances of restoration of vision are very small.

In subacute cases of glaucoma a greater reduction of tension can be effected by eserine than in the acute cases—sometimes a complete reduction. Though by the effective use of eserine in such cases the immediate urgency for iridectomy is not so great, still there can be

little doubt that the sooner it is performed the better chance there is of its proving successful.

In both acute and subacute cases of glaucoma, should the lens and iris be much pressed forward and the anterior chamber very shallow, it may be advisable to perform a preliminary scleral puncture, and, by escape of fluid from the vitreous chamber, allow of some recession of the lens. A knife can then be passed more readily into the anterior chamber and the risk avoided of splitting the layers of the cornea instead.

The recession of the lens also has the advantage of facilitating early reformation of the anterior chamber, and so preventing adhesion between the lens and cornea, which adhesion has been shown to be one cause why iridectomy may fail to relieve tension. A scleral puncture preliminary to iridectomy is strongly recommended by Priestley Smith, who has practised it extensively.

In chronic cases of glaucoma experience has shown that iridectomy is not nearly such a reliable measure for the relief of tension as in the more acute cases. It produces much the best results in the cure of the glaucomatous condition, if performed early in the disease.

Many surgeons, however, hesitate to operate on patients with chronic glaucoma when the symptoms are very slight, when the central vision is normal, and when there is only slight contraction of the field of vision. Yet it is in just such cases that the iridectomy is calculated best to arrest the disease. A very distinct objection to performing iridectomy in these cases is that the operation nearly always produces a certain amount of corneal astigmatism, so that the patient finds that the immediate effect of the operation has been to reduce his acuity of vision uncorrected by glasses, although it may be just the same as before with glasses.

The alternative treatment to iridectomy in cases of chronic glaucoma is the continued use of myotics; either eserine or pilocarpine.

Many cases of chronic glaucoma may by steady perseverance in this treatment be kept in arrest for an unlimited time. Some, in spite of it, go on steadily losing sight. Others, again, notwithstanding the myotic, as the result of some exceptional exciting or emotional circumstance, suffer an acute exacerbation of symptoms, when the surgeon is forced to operate.

The choice of treatment in chronic glaucoma, in any individual case, is a matter calling for considerable judgment and experience, it being necessary to take into consideration the patient's circumstances, age, expectancy of life, general health, and other matters.

Cases of absolute glaucoma are met with in which operative treatment of some form becomes necessary for the relief of pain. If the patient is old and feeble, or if the eye has in any way become unsightly from staphylomatous or other changes, it had best be excised. Under other circumstances the effect of an iridectomy may first be tried. In such eyes iridectomy frequently fails to produce permanent relief of tension and the pain recurs. In some of them severe intra-ocular

hemorrhage from the choroidal vessels occurs. The blood collecting between the choroid and sclerotic forces forward the retina, vitreous, and lens, the two latter structures escaping from the eye, and enucleation becoming inevitable. In rare cases this disastrous form of hemorrhage follows iridectomy for glaucoma which is not absolute.

If an iridectomy has failed in any case to relieve tension, the eye should be examined carefully to see if the lens has become tilted forward through adhesion of it to the wound. Where such an adhesion exists, the lens must be removed. In making a fresh incision the Graefe knife should be made to sweep across the posterior surface of the old one, so as to divide anything adherent to it.

If the lens has not become tilted forward, the return of tension is presumably due to a portion of the root of the iris left blocking up the filtration area. A sclerotomy should then be performed in the region of the coloboma, which will cut through the adherent root of iris, and so possibly establish a passage for fluid from the posterior chamber into the spaces of Fontana.

Sometimes in the performance of an iridectomy for glaucoma the lens is accidentally wounded and becomes opaque. This is most likely to occur when a keratome is employed and the anterior chamber is shallow, the anterior surface of the lens being pressed forward through the pupil. It has been known also to occur when the operation has been performed with a Graefe knife, from the iris having been cut through in making the upward cut.

In some cases the lens becomes cataractous after the operation, without having been wounded. In these it is generally found that there had previously been some peripheral striæ, and the manipulation of the eye acts as a maturation operation.

The operation of removal of the superior cervical ganglion of the sympathetic is one which must be regarded as still, to a certain extent, on trial. Even in skilled hands it may be attended by grave risks, and there are probably few who would care to recommend it, unless the other and simpler measures above indicated had failed.

Patients suffering from glaucoma should be advised to adopt, as far as practicable, the following habits in life: all worry, excitement, or fatigue should be avoided. They should be warmly clad and guard against anything like a chill. Great importance should be attached to their obtaining a regular and adequate amount of sleep. All straining efforts likely to lead to congestion of the head and face should, when possible, be prevented. Where there is a tendency to constipation, aperients should be administered. Errors of refraction should be accurately corrected and no very prolonged near work engaged in.

CHAPTER XII.

DISTURBANCES OF VISION WITHOUT APPARENT LESION.

By ELMER G. STARR, M.D.

The conditions described under the heading of this chapter are properly embraced by the terms *amaurosis* (loss of sight) and *amblyopia* (defective or dull sight), terms which are used to indicate all cases of defective vision which do not result from visible disease of the eye structures, and which cannot be remedied by the correction of an existing error in refraction. The diagnosis of this condition is often very difficult, as it must be based chiefly upon the evidence of subjective symptoms.

The color sense may be lost or lessened, the field of vision limited, and scotomata found. In this class of diseases may be included the following-named conditions: color amblyopia, amblyopia exanopsia, congenital, hysterical, and simulated, uræmic, glycosuric, malarial, from hemorrhage, from lightning flash, reflex, scintillating scotoma, nyctalopia, hemeralopia, erythropsia, snow blindness, micropsia, megalopsia, and metamorphopsia.

It is probable that future increase in our knowledge will remove from the category of amblyopia some of the conditions enumerated above by disclosing their real nature.

In all amblyopic conditions careful ophthalmoscopic examination should be made, as many cases of supposed amblyopia will, by careful and thorough examination, be found to depend in reality upon some disease of the retina or choroid in its extreme peripheral regions, parts of the eye which are difficult to see, and which frequently escape scrutiny in the routine ophthalmoscopic examination.

Another objective point in the examination is the macular region. This should be closely inspected by the direct method, as not infrequently in low degrees of amblyopia slight changes are found in the region of the macula, such as a granular or stippled appearance, or the presence of minute white, gray, or yellowish points—conditions which in some cases undoubtedly have their origin in long existing refractive error (eyestrain), while in others the exciting cause seems to be a renal or intestinal toxæmia.

Color Blindness. Perhaps the most wonderful of all our senses is that of adaptation or refinement of the sense of sight, the perception of color. Pure hues of red, yellow, and blue appear to the eye so positively unlike and contrasted that it is remarkable that they are

caused by waves of ether, differing only in length and rate of vibration, and that there is a graduated series of waves from one color to another, linking them together and merging the colors of the spectrum one into another.

When the sense of sight was first developed, it is probable that nothing more than light and darkness was perceived. As the visual organs became more developed, more delicate changes in light and shadow would be seen, but color, except in so far as it modified the amount of light reaching the eye, would not be visible. A high state of development of the eye as an organ of vision is compatible with the absence of all color sense, and may have existed long before the sense of color began to develop. The time at which the color sense appeared is unknown—by some it is believed to have had its origin, or at least to have developed within historic times. There are facts, however, which indicate that this sense existed in a highly developed condition in prehistoric man. Birds and many animals undoubtedly distinguish colors. Even in so low an order of animals as fishes a sense of color seems to exist, as is evidenced by their protective coloring. It is quite possible, of course, that the color sense of the lower animals may not be identical with that in man, but a fact which points to the early development of this sense is that babies have a well-developed sense of color, which would hardly be the case were this a recent acquirement of the human race.

Color has no objective existence, but is an internal sensation, and may be caused by pressure on the eyeball or any means which stimulates or excites the retina of the eye. In the present chapter it is considered as due to the action of light waves.

Objectively, then, color corresponds to light waves or ether undulations of certain length and rate of vibration, at least this will apply to such colors as have a known vibration for their cause. There are colors, however, which have no objective definite ether waves for their production, and which serve to illustrate still further the fact that color is purely a physiological sensation. Such colors are purple, which does not occur in the spectrum, and has no definite wave length for its production; and white, if this may be called a color. By experiment it has been determined that the sensation of red is caused by the longest visible wave and slowest rate of vibration, while the shortest wave and the most rapid vibration give the sensation of violet. Red, then, gives us one end of the visible spectrum, and violet the other end. Between these limits there is a graduated series of wave lengths, all of which affect our visual apparatus and give us the various colors of the spectrum. The spectral colors pass from one into another by such slight gradations that, when examined in a long spectrum, no sudden change from one color to another is found to occur, but one merges so gradually into another that it is difficult to say just where one color ends and another begins, so that the spectrum is found to be made up of an infinite number of gradations of colors. If, however, the spectrum is made shorter, so

that the colors are more condensed, as it were, it appears as if made up of only three or four colors—red, green, blue, and violet, and the transition from one color to another is more abrupt.

There are several theories to explain the manner in which the different colors affect the eye. Without entering into a discussion of the subject here, it may be stated that in general these theories suppose the eye to be provided with sets of sensitive elements which are affected either directly or secondarily by some three or more of the spectral colors. For, while the decomposition of white light by means of a prism gives seven prismatic or spectral colors, it is found that all these colors, as well as white, may be obtained by combining three colors, such as red, blue, and green.

In the Young-Helmholtz theory the retina is supposed to be provided with three sets of elements, one set of which responds most strongly to red rays, another to green, and a third is most affected by blue light. All the elements are, however, affected to some extent by each of the three colors mentioned. Thus, red light exerts its greatest action upon the red-sensitive elements, although it affects the green also, and to a lesser degree the blue-percipient elements. Similarly with green and blue, all the elements are affected, but in varying degrees. The simultaneous action of red, blue, and green gives the color or effect of white light.

The effect of color waves upon the percipient elements is supposed to be due, not to the action of light waves directly, but to the decomposition which they cause of a photo-chemical substance with which the sensitive retinal elements are supplied. That is, the red-sensitive retinal elements are affected by the decomposition of a photo-chemical substance which is most sensitive to the red rays of the spectrum. Similarly the green-sensitive and blue-sensitive elements are affected by green and blue light waves.

Impaired color sense, or color blindness, exists in 3 or 4 per cent. of males, and is less common in females. Color blindness may be total or partial. Totally color-blind individuals see the spectrum in different shades of gray, and all objects appear to them much as they do to normal eyes in stereoscopic photographs. To the partially color blind the spectrum appears in two colors only, with a gray or neutral band in it. The most common forms of color blindness are red- and green-blindness. These are sometimes classed under one head, viz.: "red-green" blindness, from the fact that the red-blind do not see green correctly, and the green-blind do not see red correctly—in fact, are blind to both colors. There are two classes of "red-green" blindness, and there is a clinical difference between them. In one class the spectrum is shortened at one end (the red), while in the other the spectrum is not shortened, but has a neutral zone in it. Those having this defect see some colors correctly, other colors incorrectly, and the rest, those to which they are "blind," not as colors at all, but as neutral grays. Bearing this in mind, the following schedule from Le Conte

will help make intelligible what the color blind see, what mistakes they are apt to make in matching colors, and the means adopted in detecting this defect:

PURE COLORS.

I. *See Correctly.*
- *a.* White and black and all intermediate shades, or grays.
- *b.* Yellow and all shades of the same—*i. e.*, brown.
- *c.* Blue and all shades of the same or slate blues.

II. *Do not see at all as colors.*
- *a.* Reds are seen as different shades of gray.
- *b.* Greens are seen as different shades of gray.

MIXED COLORS.

III. *See incorrectly.*
- *a.* Scarlet, which is a mixture of red and yellow light, is seen as gray and yellow, which equals dark brown.
- *b.* Orange = red + yellow, are seen as gray + yellow = lighter brown.
- *c.* Purple = red + blue, are seen as gray + blue = slate blue.
- *d.* Yellowish green = yellow + green, are seen as yellow + gray = brown.
- *e.* Bluish green = blue + green, are seen as blue + gray = slate blue.

To be clinically accurate, this table should be modified in some ways, inasmuch as it does not distinguish two classes of red-green blindness—one with and one without shortened spectrum. The table serves its purpose, however, as an aid in elucidating the subject. From this it will be observed that the red-green blind are very liable to confuse or mistake all mixed colors, as well as reds and greens with either browns or gray blues.

Blue blindness is rare and of little importance clinically. These three types comprise practically all cases of color blindness, although there are many deviations from the general types.

One curious result of color blindness is that persons having this defect are able to discriminate between certain hues which to the normal eye appear identical: *e. g.*, two complex solutions may have the same color to the normal eye, but to the color-blind eye some one or more of the chromatic constituents of the solutions may not be perceived, and in consequence the two solutions appear to differ in color.

Color blindness is usually a congenital defect, but it may be an acquired condition, depending on some disease process involving the retina, optic nerve, or visual centres, such as atrophy of the optic nerve, tobacco amblyopia, and cerebral injuries or disease. In the acquired form the color blindness may be limited to a part of the visual field, either peripheral or central. Another difference between the congenital and the acquired forms of this defect is that in acquired color blindness the acuteness of vision usually is lowered, while in the congenital form this is not the case.

TEST FOR COLOR BLINDNESS. Of all tests—and there are more than forty different ones—the wool test of Holmgren is probably the one most frequently used. The set of wools consists of a selection of worsted yarns dyed with various colors. The skeins of test-colors

are three in number, viz.: green, rose pink or purple, and red. Of the remaining skeins, some have the same color as the test-skeins, while others are dyed with mixed or "confusion" colors. A colored plate accompanies the set, and is used as an aid in deciding the character of the color blindness, and is not to be used in any way as a test-object.

The test is made in good light—daylight, if possible—and the eyes are tested separately.

The wools are placed in a heap on a white or black cloth covering a table, and the test-skein of green is placed at one side, separated from the pile, and the person under examination is requested to select from the pile all other skeins resembling the test-skein in color, but not to attempt to match it in every respect, as no two skeins in the pile are exactly alike; all shades of the same color are to be selected. If in the test the person examined selects some of the confusion colors, such as light brown or gray, as well as different shades of green, then he is shown to be color blind, and if the existence of color blindness alone is to be determined no further test is required; but if the kind of color blindness is to be determined, examinations with other test-skeins should be made. The second examination with the purple test-skein will show that "red-blindness" exists if the colors which are selected to match the purple include shades of blue or violet; while if green or gray is selected the subject is "green-blind."

Dr. William Thomson has devised a very convenient modification of the Holmgren wools, which consists of a stick with yarns attached, as shown in Plate XIX. As in Holmgren's method, the test-skeins are green, purple, and red. The test-skeins are to be matched in turn from the colors on the stick, which are arranged in alternate match and confusion colors, and which are numbered from one to twenty. Ten tints are to be selected.

The odd numbers being the match colors and the even numbers the confusion colors, it is evident that the selection made by the color-blind eye will include some of the even numbers, while the eye free from this defect will select only odd numbers.

Dr. Thomson has further improved his test by discarding the color-stick and increasing the number of the color-skeins to forty, each of which has a bangle attached bearing a number. By this means the skeins may be heaped together without any regularity of recurring colors, such as occurs on the color-stick, thus obviating the possibility of giving a hint to the person under examination by the regular arrangement of yarns, as might occur after repeated examinations of the individual.

The Holmgren test is very satisfactory in detecting congenital color blindness, but in the acquired condition some other method must be used, because of the fact, before mentioned, that the blindness may be limited to a small area, so that the color of a large object is correctly seen, and the defect in the color sense is discovered only when using a small test-object, such as a distant signal light, whose retinal

PLATE XIX.

image is so small as to fall entirely within the color-blind area. It is, therefore, evident that should the wool-test indicate no defect in the color sense, we cannot be sure without a further examination that a color scotoma—a tobacco scotoma, for example—does not exist.

The existence of central color scotoma may be detected by the use of the perimeter, employing small squares of colored paper of from one to ten millimetres size.

A more satisfactory method is to use as test-objects small distant colored lights, the different colors of which should be distinguished; or a dark-blue cobalt glass is placed over each eye separately and the vision is directed to a small distant light, such as the flame of a candle. A normal eye sees a light of one color surrounded by a halo of another color, but a color-blind eye sees but one color, blue, or a white light surrounded by a blue halo.

Individuals who are blind to red are also blind to its complementary color. Application of this fact as a test for color blindness may be made in the following way: A sheet of white paper is illuminated by two lights placed at a little distance, one light having a red glass in front of it. Between the lights and the paper a small object, such as a pencil, is placed so situated that two shadows of it fall on the white paper. One shadow, that formed by the rays from the white light, will be red in color, while the other shadow will be its complementary color, or green. A color-blind person will see but one shadow under these conditions; or at least will distinguish no difference in their colors, but merely a difference in the density of shade.

Amblyopia ex Anopsia. *Amblyopia from Disuse.* If in early childhood an eye is disused, its visual power is diminished from imperfect physiological development of the central visual centre. An active factor in causing this condition is probably the act of suppressing the vision in the disused eye. Particularly is this true if, as usually occurs in strabismus, the vision of the squinting eye is suppressed because of the confusion of images otherwise seen.

This suppression of vision, which really means an abeyance of the physiological processes in the visual centres, together with the youth of the patient, is undoubtedly the important factor in the production of amblyopia exanopsia.

Age also is important in determining the result of disuse. In adult life, after the visual centres become physiologically developed, amblyopia does not result from disuse. It is in the first years of life, the years of developmental activity, that disuse of this function, coupled with efforts to suppress it, exhibits its effect. Thus it will be evident that in all cases of squint in children efforts should be made to maintain physiological activity in the squinting eye by exercising its functions for a short or long time daily, while the other eye is excluded from work by means of a bandage, patch, or opaque glass. The amblyopia accompanying squint is, however, frequently, if not usually, a congenital condition, and in no way the result of disuse.

But in cases of squint, even in those having normal visual acuteness in each eye, the power of binocular fixation becomes seriously impaired, so that true binocular fixation is rarely attained after correcting a strabismus.

In cases of high refractive error, not infrequently a considerable degree of amblyopia remains after correction of the refraction by suitable lenses. In a certain number of these cases the acuteness of vision improves when correcting-glasses are worn, at first rapidly, then more slowly, until vision becomes normal, or the condition may become stationary before normal vision is attained.

Congenital Amblyopia. This is probably the result of arrested or imperfect development of the central visual centre. It often escapes observation during childhood, and, indeed, not infrequently is undiscovered until adult age or middle life, when by chance, perhaps, the patient discovers that one eye is blind.

When affecting one eye, it is associated frequently with strabismus. Nystagmus, also, sometimes accompanies high degrees of amblyopia. The entire field of vision may be affected, or scotoma may be present. No objective conditions adequate to account for the defective vision are found. The condition is not amenable to treatment.

Hysterical Amblyopia. Among the ocular manifestations of hysteria, amblyopia is often present. It is always associated with concentric contraction of the field of vision. A peculiarity of this form of contracted field is that it may be modified in size by excitation of the skin. A puncture of the skin by a pin, for instance, will increase the size of the field, and by successively irritating or exciting the skin, the field may be increased to its normal size.

Color sense is often disturbed, and there may be inversion of the normal color field—*i. e.*, instead of the field for blue being largest and that for green the smallest, the field for green is found largest and that for blue smallest. There may be polyopia, monocular diplopia, micropsia, megalopsia, hemianopsia, or central scotoma.

The acuteness of vision is often improved by plain blue glasses.

When blindness is complete, it is usually monolateral, and the pupil reacts when the other eye is covered and uncovered.

Other disorders of sensation, particularly of the mucous membranes and skin, and especially of the pharynx, almost always accompany hysterical amblyopia.

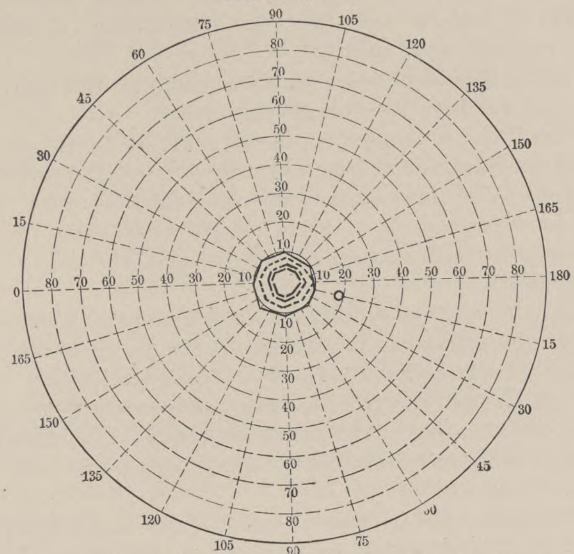
Simulated Amblyopia. Blindness, complete or partial, affecting one or both eyes, is sometimes pretended by individuals who hope thereby to escape the performance of some duty or to gain compensation in the way of a pension, or damages. If the individual is intelligent and clever, or, on the other hand is densely stupid, it may be very difficult to convict him of deceit, and much care must be given to the examination of his condition.

Complete blindness of both eyes is not often simulated, except in those cases where a considerable degree of amblyopia really exists. In these cases the action of the pupil is, perhaps, the best index of the

condition. In absolute blindness in both eyes the pupils will rarely respond to light. Should the patient have used atropine to

FIG. 340.

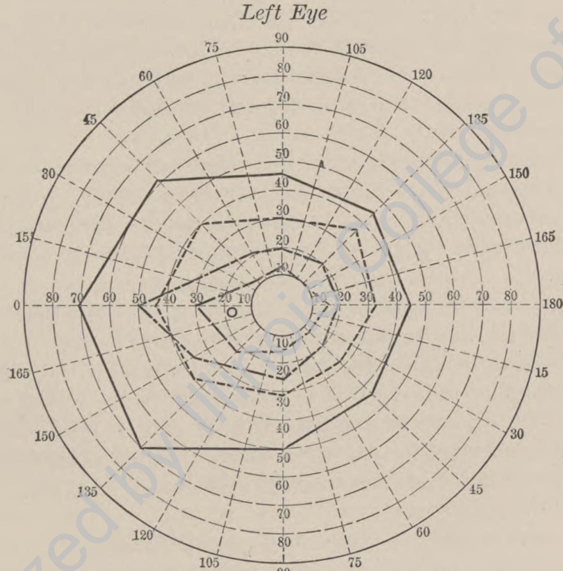
Right Eye



Extreme concentric contraction as produced in hysteria.

FIG. 341.

Left Eye



Reversal of field as seen in neurasthenia.

mask the real condition of the pupils, this fact may be suspected if there is extreme dilatation, as the mydriasis accompanying amaurosis is usually moderate. It is true, however, that the pupils may contract under the stimulus of light, or that the opposite condition, that of wide dilatation of the pupil, may either of them accompany blindness; so that it will be seen that the unmasking of malingering in these cases may be very difficult.

A close watch should be kept on the actions of the individual when he believes himself free from observation.

A prism of 6° or 8° may be placed with its base out before one eye and left in place for a few minutes. If on its sudden removal the eye is seen to deviate outward, it may be suspected that the eye sees. Should repeated tests show that withdrawal of the prism is accompanied by this movement of the eye, the fact that the eye sees is established.

If blindness in one eye is claimed, a prism placed before the pretended blind eye will cause no change in the position of the eye if it be really blind. If, however, the eye does participate in vision, a prism of 6° or 8° placed with its base out before the eye causes the eye to move inward, and if the prism be suddenly removed while the vision is fixed on some near object, the eye can be seen to move outward.

Prisms strong enough to cause double vision may be put on the patient, and he be requested to walk or to go up and down a few steps of a flight of stairs.

The diplopia caused by a 5° or 6° prism with base up or down, for example, is most confusing. In making these tests, it is, of course, important to see to it that the patient does not close the "blind" eye. With a 6° or 7° prism with base down in front of one eye, and the vision directed to some distant object, the eyes will alternately move up and down if they be alternately covered and uncovered with a screen.

A prism may be held with its thin edge opposite the middle of the pupil of the seeing eye, thus causing double vision in the single eye. When the patient's attention is directed to the fact that he can see double with one eye, the prism may be moved so as to cover the pupil, when if he still admits that double vision exists, he is seeing with both eyes. These tests show the existence of binocular vision, but do not indicate the acuteness of vision in the blind eye.

Among the quantitative tests may be mentioned the following: A strong convex lens—*e. g.*, 10 D.—is placed before the seeing eye, and the test-type is held at such a distance that it can be read with this eye, which with an emmetropic eye is one-tenth metre; then with both eyes open the type is moved farther away, and if it is still read, it is being read by the "blind" eye.

The stereoscope may be used with cards bearing various designs which differ on the two sides, and which are combined by the vision of two eyes to form a definite figure or letter.

Before the seeing eye two strong cylinders—*e. g.*, 4 D. and +4 D. —are so arranged as to neutralize each other, *i. e.*, with axes together. In this position they do not affect the vision. Now, while the patient is reading distant test-type one cylinder is turned 10° or 15°, thus entirely changing the refraction of the combination and lessening the visual acuity of the eye before which it is placed. This change should be made while the patient is reading large type, as in reading small type, which demands of the patient close attention to detail, a comparatively small change in the lens before one eye is noticed at once, even though both eyes have normal visual acuteness and are being used together. When one eye has subnormal vision, then any change in the refraction of the lens in front of the better eye is, obviously, more quickly detected.

Of course, the refraction of the eye should be determined, at least approximately, by using the shadow test with the supposed blind eye, and efforts to improve vision by correcting lenses should be made.

Snellen's transparent red and green test-letters of different sizes may enable one to detect simulation, and at the same time to determine the amount of vision in each eye. The patient is requested first to read the letters without anything before his eyes. Then a spectacle frame, holding a red glass in one side and in the other a green glass, is put on him, and he is asked to read the letters again, taking care that he keeps both eyes open. The green glass shuts off from one eye all the light coming from the red letters, thus making them invisible to the eye. Similarly the red glass makes the green letters invisible to the other eye. By noting what letters are read, it is easy to determine whether one only or both eyes are used, and what acuteness of vision is represented by the size of type read.

Uræmic Amblyopia. Amblyopia from uræmic poisoning is seen accompanying the albuminuria of scarlet fever, variola, measles, and pregnancy. It is associated frequently with symptoms of brain irritation, such as vomiting, convulsions, coma, and hemiplegia. Both eyes are affected, and blindness may be complete within a few hours from its onset. A peculiarity of this condition is that the pupillary reactions are not lost. The prognosis so far as vision is concerned is good, blindness usually disappearing with subsidence of the albuminuria.

Glycosuric Amblyopia. Diabetes sometimes causes amblyopia, and it is characterized by a central color scotoma. Central scotoma for white may also be present. The visual field may be normal or contracted, or may be hemianopic. The prognosis is unfavorable, although useful vision may long be retained.

Malarial Amblyopia. Malaria is another disease which, in addition to those cases of impaired vision due to apparent lesion, causes other disturbances of vision in which the ophthalmoscopic findings are negative. The affection appears as a transient loss of vision, lasting from a few hours to several days, and disappears under treatment with quinine. The amblyopia begins with the chill and ends

with the onset of the sweating stage. Although blindness may be complete, the pupils react normally to light.

Amblyopia from Hemorrhage. Loss of blood is followed occasionally by impaired vision or by blindness. The disturbance in vision may not manifest itself until some time after the hemorrhage, even after the normal volume of blood has been re-established and the equilibrium of the circulation restored. Instances of this condition have been observed following hemorrhages which were not very severe or exhausting, so that a condition of anæmia could not be said to exist, although it more often accompanies or follows an exsanguinated condition. It is seen after metrorrhagia, hæmoptysis, gastric hemorrhage, and post-partum hemorrhage. The amblyopia sometimes accompanying great anæmia and extreme chlorosis may be considered the same in kind as that following hemorrhage. A case of this nature, under the observation of the writer, occurred in a frail, anæmic woman, thirty-six years old, after a prolonged period of lactation. Vision gradually failed for several days, when blindness became absolute. The light reaction of the pupils was not lost, but was lessened, and the pupils were but little larger than normal in size. Tonic treatment was at once instituted, and the child was taken from the breast. The condition of vision remained unchanged for three weeks, when sight began to return, and soon normal vision was restored.

The loss of vision may be gradual, or sudden and complete, or partial, and is generally bilateral. There may be central scotoma. The affection may remain for a few hours or days, or even for a few weeks, and then gradually disappear completely, or it may leave behind it permanent defects. The most unfavorable cases appear to be those which are late—a week or more—in following the hemorrhage, and these cases usually show, at a later stage, atrophy of the optic nerves.

It should be remarked that an impairment of vision coming on during an exhausted bodily condition may be due to a weakening or exhaustion of the power of accommodation, and the state of the refraction and accommodation should always be examined in such conditions.

It is, of course, important to have some guide as to the prognosis in these conditions. The negative result of the ophthalmoscopic examination is no index in making a prognosis, for we meet with cases of sudden blindness occurring during the course of acute disease, in which no change in the eye fundus is visible for weeks, and yet where blindness is permanent and in which atrophy of the optic nerve eventually appears. The behavior of the pupil may give information of value, as when pupillary reaction is not lost in blindness in any of the foregoing conditions the prognosis is more favorable.

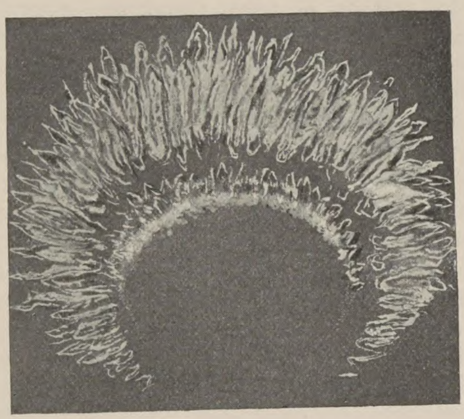
Amblyopia from Lightning Flash. Loss of vision by lightning stroke has been reported in many instances, and may be accompanied

by other lesions, such as burn of the skin or cornea, ptosis, or injury to the lens, causing cataract, or inflammatory conditions of the deep structures of the eye. The sight in cases uncomplicated by inflammatory or other visible changes is usually fully restored.

Reflex Amblyopia. Irritation of the fifth nerve, especially that form arising from diseased teeth, is said sometimes to affect vision. Amblyopia in one eye, resulting from irritation of the ciliary nerves in the other eye, has been reported, as have cases of amblyopia from intestinal irritation due to the presence of worms in the intestinal canal.

Scintillating Scotoma. This condition (Fig. 342) is known also as "amaurosis fugax," and as "flicker scotoma." The condition is characterized by the appearance in the field of vision of a cloud, obscuring more or less completely objects in one part of the field;

FIG. 342.



Scintillating scotoma in migraine. (REUTE.)

this cloud increases in size and may obliterate completely one-half of the field. It is homonymous—*i. e.*, affects the nasal half of one visual field, and the temporal half of the other. Accompanying this cloudiness is a peculiar wavy or flickering visual sensation. Often sparks of light appear, or the cloud may be bordered by a bright edge. The acuteness of vision is disturbed, being generally very decidedly decreased. The scintillations finally cease, the cloud disappears, and vision is again normal. Accompanying the sensations just mentioned, or immediately following their subsidence, headache usually appears and is limited to one side—migraine. These phenomena may result from irritation, probably of the brain cortex, from toxic substances absorbed during gastric or intestinal indigestion. Eyestrain from refractive errors is surely a not infrequent cause, either directly by causing brain irritation, or indirectly by reflexly disturbing the functions of the stomach.

As temporary obscurations of vision occur in glaucoma, the tension of the eyes should always be examined in this condition, in order to avoid mistaking the character of the affection.

Nyctalopia.[1] Night blindness is a functional disease characterized by a diminished sensibility of the retina to light. It usually occurs suddenly in spring or summer, after one or several days spent in bright sunlight, a debilitated condition of the health often being a contributing cause. In the beginning the centre only of the retina is affected, and a more or less sharply defined cloud appears in the centre of the field, which compels the patient to look beyond or at the side of an object in order to see it at all distinctly. Frequently all of the field, or all but the periphery, is cloudy. If the exciting conditions continue, the density of the cloud becomes greater, and the blindness comes on earlier in the evening. In cases of only moderate severity the full light of a bright or even a cloudy day is sufficient to permit the patient to read or distinguish objects near at hand. In high degrees of night blindness, however, very bright illumination is necessary for normal vision. An unfavorable position of the object to the light, a cloudy day, shadows falling on objects, all materially lessen the visual acuteness, and lessen the power of distinguishing colors. Sudden changes in illumination affect the vision much more than is the case with the normal eye. If the illumination is reduced, a point is reached beyond which the decrease in vision is very rapid, so that a very small decrease in illumination causes a profound effect upon vision, often rendering objects instantly unrecognizable; even perception of light may disappear. This particular point in decreased illumination varies with different individuals. Diminished light at any hour of the day has the same effect, and the idea, at one time common, that the disease was confined to certain hours of the day, is incorrect. The pupils are usually somewhat dilated, but react to light, and the field is often irregularly contracted. The color sense is frequently disturbed, and colored vision may exist.

A cure usually follows removal of the cause, but relapses are frequent. Protection of the eyes against bright light and the use of tonic medicines comprise the treatment of this affection.

Hemeralopia. Day blindness is a form of retinal hyperæsthesia in which vision is diminished during ordinary daylight, but is good in a dim light. It may be caused by exposure to bright light, especially by light reflected from glistening snow or ice. An important contributing cause is eyestrain from refractive errors. Intense photophobia is sometimes present, as well as the phenomena called phosphenes, which consist of subjective symptoms characterized by the appearance of highly luminous moving clouds, rings, or streaks, and dazzling sensations. The condition may exist as one of other

[1] The term nyctalopia is often used to indicate day blindness, and hemeralopia to indicate night blindness. The true meaning of the words, however, seems to be as above used.

congenital defects, such as albinism or coloboma of the iris or choroid.

The use of tinted glasses, but especially the careful correction of refraction, will eventually cure the affection.

Snow Blindness. This may take the form either of day blindness or night blindness, and follows exposure to the sun's rays reflected from snow fields. It is accompanied often by inflammation of the conjunctiva or cornea, intense photophobia, and spasm of the lids, although it may not be accompanied by inflammatory conditions. It begins by a gradual or rapid darkening of the visual field, and continues as long as the eyes are exposed to glaring light. Protection of the eyes against the bright light by means of smoked-glasses, or otherwise, relieves the condition.

A condition somewhat similar to the foregoing is met with sometimes in persons who are employed in the care of the electric arc light, the intense light of which has an injurious effect on the eye structures.

Micropsia, Megalopsia, Metamorphopsia. In micropsia the condition of vision is such that objects look too small; in megalopsia they look too large; and in metamorphopsia they appear distorted.

One or all of these conditions may be present when the retinal rods and cones are displaced by exudate or other cause. If the retinal elements are pushed asunder, so that the images which fall on the retina cover fewer elements, the objects seen will appear smaller than they are; if the retinal elements are pressed together, the opposite condition exists, and it is evident that distortion of objects may appear from disturbance of the order of the rods and cones. These conditions of vision are made out best by causing the patient to look at a set of parallel lines drawn on a surface and held near at hand, when, if the central lines appear bent toward each other at the point of fixation, micropsia with metamorphopsia appears, while if the lines seem bent apart at this point, megalopsia exists.

Paresis of, or weakened accommodation, whether resulting from disease, or produced artificially by drugs, has the effect of causing near objects to appear smaller than is natural. The psychic effect of the increased effort of accommodation required to see the object distinctly is to give the impression of a much smaller object. In hysteria objects sometimes appear too large or too small.

Metamorphopsia may be caused by irregularities in the curvature or density of the refracting media of the eye.

Erythropsia. This is a condition characterized by saturation of the field of vision with a certain color, and may be due to coloring matter in the dioptric media or anterior layers of the retina. It is sometimes seen during the occurrence of icterus, and is then probably due to the presence in the eye structure and fluids of bile pigments. Colored vision is often noted after cataract extraction, in which case the color of the field is complementary to the color of the light which the eye saw through the cataractous lens. That is, the color of a

cataractous lens is usually yellowish, and it transmits light of this color, so that after the yellowish lens is removed the eye sees white light as bluish in color. This condition disappears in the course of time and demands no treatment. Red vision sometimes follows exposure of the eyes to strong light.

Gazing at the sun through a telescope having a colored glass behind the eye-piece is followed by colored vision persisting for days, the color seen being complementary to the color of the glass behind the eye-piece. The ingestion of certain drugs, such as cannabis indica, santonin, amyl nitrite, picric acid, osmic acid, and some others is often followed by colored vision. Coffee is said sometimes to cause red vision.

CHAPTER XIII.

THE EYE IN ITS RELATION TO GENERAL DISEASES.[1]

By C. F. CLARK, M.D.

CONSTITUTIONAL DISEASES.

Anæmia is secondary to so many and such diverse pathological conditions that, in considering its relation to diseases of the eyes, it is necessary to observe caution to avoid confusing the effects of the anæmia proper with those of the disease upon which it depends; and, even if we exclude the primary or essential anæmias (chlorosis and idiopathic or progressive pernicious anæmia), there is, probably, no one constitutional condition which more frequently has a part in producing the various forms of asthenopia, the consideration of which occupies so much of the time of the ophthalmic surgeon.

No subject pertaining to the complex relation which exists between the eye and general diseases can be of greater importance to the physician than that which concerns the functional reflex disturbances so often seen in patients who have errors of refraction and are at the same time more or less anæmic.

There exists little doubt in the mind of the writer that the early and judicious use of those means, hygienic, dietetic, and medicinal, which tend to overcome anæmia, could, in a large number of instances, delay for many years the necessity for correcting the low grades of hyperopia and astigmatism which are so important a feature in the practice of ophthalmology as we see it in America.

The general asthenia which accompanies the anæmic state manifests itself as asthenopia, and this may be conjunctival, ciliary, muscular, or retinal. It not infrequently happens that after a prolonged struggle on the part of the ophthalmic surgeon to correct properly hyperopia, astigmatism, and muscular imbalance by means of spherical, cylindrical, and weak prismatic lenses, and to relieve obscure reflex symptoms, such as headache, etc., apparently due to eyestrain, and obstinate photophobia and conjunctival irritation, by appropriate treatment, it is found that large doses of a ferruginous tonic and a properly regulated life, with an abundance of out-of-door exercise, bring about complete relief from all the distressing symptoms, rendering glasses for the time being unnecessary. On the other hand,

[1] The author wishes to acknowledge his indebtedness to Dr. W. K. Rogers for the valuable assistance afforded him in collecting material for the following chapter.

it frequently happens that all of the best directed plans of general treatment completely fail when they are not supplemented by the most painstaking correction of all such errors of refraction and muscle imbalance.

An ideal life is seldom possible to our patients, and the practical problem which faces the general practitioner, as well as the ophthalmic surgeon, in dealing with such cases is, How shall we afford relief to the symptoms of which they complain, and still allow them to continue to live the life and follow the pursuits which seem necessary to them? Under these circumstances it is essential not only to correct all errors of refraction and imbalance, but also to treat the anæmia which renders them a more active source of disturbance.

In simple anæmia, even when profound, the ophthalmoscopic appearances are often negative. In some cases we find pallor of the disk and under-filled bloodvessels. When sufficiently prolonged to produce wasting, the eyeball may become somewhat sunken in the orbit.

Congestion of the conjunctiva accompanied by dryness is sometimes noted, and it is not uncommon in the writer's experience to find cases of what would be classed as palpebral conjunctivitis, in which there is a sensation of the presence of dust particles in the eyes, yield only after the anæmia has been relieved by treatment.

There are accidental conditions that may give rise to a number of eye symptoms in well-marked cases, even when not of such a character as to justify the term pernicious anæmia—œdema of the lids, extravasation of blood appearing suddenly, generally at night, beneath the bulbar conjunctiva, and, at times, even small retinal hemorrhages.

In speaking of pernicious anæmia, Knies, quoting Fraenkel,[1] mentions parenchymatous changes in the external ocular muscles. They were pale and clay-colored, with partial absence of the transverse striations, and the fibres were filled with a yellow or brown pigment, or were finely granular, some fibres being narrow and waxy.

Occasionally in this condition we may have even neuritis or retrobulbar neuritis, followed by atrophy of the optic nerves. In chlorosis eye symptoms present themselves of a character similar to those of anæmia; but, unlike pernicious anæmia, chlorosis is not accompanied by retinal hemorrhage.

Leukæmia. In this fatal disease eye symptoms may or may not be present. In the acute stage hemorrhages into the lids or conjunctiva may appear, and they are seen also at times as a late manifestation. Neoplasms of leukæmic origin may develop in the orbit, though this is an extremely rare occurrence, and involvement of the lacrymal glands and lids has been reported. Occasionally the iris and choroid are involved; in the former, circumscribed tumors sometimes appearing before the lymphatic and splenic enlargements, and

[1] Deutsche Arch. f. klin Med., xx.

certain cases present the appearance of chronic iritis with flocculent opacities of the vitreous (Berger).

Hemorrhages and exudations into the choroid and optic nerve have also been observed, although the most frequent seat of such lesions is the retina, where one may sometimes see bright areas of degeneration. Poncet has shown how vascular degeneration makes hemorrhage possible, and de Schweinitz describes the white spots with red borders which make their appearance in the macular region and also near the equator. These spots are said to consist of leucocytes surrounded by red corpuscles. "In some cases the orange-red color of the fundus is masked by a fine, striated, grayish veil, due to opacity of the superficial covering of the retina" (Berger).

Although albumin may be present in the urine in leukæmia, the bright white areas of infiltration need not mislead one who takes into account the other general manifestations of the disease, especially the microscopic appearance of the blood.

Rhachitis. Whether due to rhachitis or some other concomitant dyscrasia, interstitial keratitis and phlyctenular conjunctivitis and keratitis are observed in rhachitic subjects. Lamellar or zonular cataracts, either congenital or forming in early childhood, in which we find alternate layers of opaque and transparent lens tissue, depend for their development upon constitutional diseases which interfere temporarily or periodically with the nutrition of the lens, and their formation is analogous to and at times associated with corresponding interruptions in the formation of the enamel of the teeth.

Hereditary syphilis, scrofula, and rhachitis have all been assigned as causes of this interference with the regular process of development, but it is to the prolonged and violent convulsions accompanying the last-named disease, and occurring during the period of active development of the lens cells, that the majority of authorities attribute this form of cataract. While quite generally accepted, considerable doubt is thrown upon this theory, so far as the convulsions are concerned, by the fact that prolonged infantile convulsions so frequently occur without the development of cataract, and the additional fact that in so large a proportion of cases no history of convulsions can be elicited.

Hæmophilia. This condition, so little understood, causes disease of the eye far less frequently than would be expected. Priestley Smith has reported one case of orbital hemorrhage following an injury in a "bleeder," and Haab states that retinal hemorrhage in the form of retinitis proliferans has been observed. Surgical procedures in such cases are to be avoided when it is possible, and especially those involving the use of the knife.

The writer has removed without accident a papilloma from the conjunctival sac by means of a ligature in a pronounced hæmophiliac.

Addison's Disease. Aside from the asthenopia which naturally accompanies a disease characterized by marked general weakness, the eyelids may share in the general bronzing of the skin of the face, and

there is apt to be jaundice of the conjunctiva, while Schroetter[1] saw patches on the sclera.

Myxœdema. This disease may make its appearance first in the skin of the eyelids. Amblyopia has been reported, and Wadsworth saw one case with atrophy of the optic nerve involving both eyes.

With general alopecia there is falling of the eyelashes, and this as well as the failure of accommodation and concentric limitation of the visual field, which may occur without apparent atrophy of the optic nerve, has been known to improve or disappear under treatment with thyroid extract.

Diabetes. *Diabetes Mellitus*. Probably no other constitutional disease produces a greater variety of ocular manifestations than diabetes, and yet in its milder forms it may be present for a long time without evidence of its existence being apparent in the eyes. In temporary toxic and traumatic glycosuria, notwithstanding the presence of large quantities of sugar in the urine, the eyes are not affected (Knies), which would tend to confirm the theory that the presence of the sugar is not the direct cause of many of the symptoms.

Among those who support the various theories of the etiology of this interesting disease there seems to be a general agreement on one point, and that is, that the underlying cause is some profound disorder of the nervous system.

Undoubtedly many of the ocular manifestations at times attributed to diabetes are accidental or indirect effects; but when it is so far advanced that assimilation is seriously interfered with and evidences of auto-intoxication present themselves, we find, as in albuminuria, the most profound changes in almost every portion of the eye, and, while it is to the condition of the crystalline lens and retina that attention generally is directed, the external ocular muscles, the cornea, the iris and ciliary body, the lens, the vitreous and the choroid, retina, and optic nerve may all be affected.

In advanced cases we may have a somewhat intractable form of eczema of the edges of the lids, and there is also a tendency at times to the formation of furuncles.

Many instances have been recorded of diabetic paralysis of the external ocular muscles, any of which may be affected; and Leber has pointed out that such paralysis may be due directly or indirectly to diabetes or may result from the cerebral disease on which it depends. While some authorities state that paralysis more commonly affects the branches of the oculomotor, Hirschberg and Lawford agree that in their experience the sixth is affected more frequently. Unilateral ptosis, from paralysis of the third, and lagophthalmos from involvement of the facial, are seen occasionally.

The paralysis may in the more advanced stages be permanent; or it may be slight and temporary in character in those cases in which the constitutional malady yields to treatment. Nuclear and periph-

[1] Wien. med. Blätt., 1886, No. 21.

eral hemorrhages and toxic peripheral neuritis have been assigned as the causes of paralysis of the external ocular muscles, and to the latter cause Knies assigns the frequent neuralgias and occasional anæsthesias and sensory disturbances which are seen in diabetic patients. He calls attention also to the fact that a peripheral neuritis of diabetic origin may cause herpes zoster ophthalmicus, and that the anæsthesia of the first branch of the trigeminus may give rise to neuroparalytic keratitis.

As in other diseases causing marked impairment of nutrition, diabetes in its terminal stages sometimes causes destructive keratitis.

Diabetic iritis is by no means uncommon, as was established by Leber, in 1885, and has been confirmed by many writers since that time. Hutchinson states that in his experience it generally occurred in patients who were also the subjects of gout. As a complication of operations, such as cataract, diabetic iritis is not only very intractable, but also not very uncommon. Iritis in diabetes is generally of the plastic type; but, with the exception of cases following operations, is not usually severe. When there is exudation it is generally fibrinous in character, and may entirely block the pupil, in some instances being associated with hypopyon and hyphæmia. Cyclitis is seen occasionally, and is followed by degenerative changes in the vitreous.

That cataract occurs as a result of diabetes is conceded by all authorities, but there is some difference of opinion as to how it is produced. When occurring in elderly subjects of diabetes the possibility of its being a mere coincidence should be borne in mind; but while it may occur at almost any age, diabetic cataract is often seen in quite young people, several instances being recorded in patients ranging in age from eleven to fifteen years.

The proportion of individuals with diabetes who develop cataract has been variously estimated at from 4 to 25 per cent., the latter being the result of von Graefe's observations.

It is questionable whether it is ever possible to distinguish by a physical examination between a cataract due to diabetes and one due to other causes, although, as they often appear in relatively young people, they are apt to be soft, usually developing rapidly, and are at times preceded by almost visible swelling of the lens. They may appear in cases in which the general nutrition has been only slightly affected as well as in those in whom there is great emaciation; and there is great difference of opinion as to the mode of development. The majority of the elaborate theories advanced to account for the production of cataract in diabetes are easily proved to be unworthy of consideration, and this applies especially to the theory that the opacity is in some way due to the chemical effect of the sugar which is found in the substance of the lens. This theory proves too much, for sugar has been found in the lens in two-thirds of the cases of diabetes, and still more frequently in the aqueous humor and vitreous. It is probable that Knies is correct when he states that diabetic cataract

develops under the same conditions as spontaneous cataract, as the result of disturbances in the choroid, and particularly in the ciliary processes, which furnish the nutritive supply to the lens. Toxic substances circulating in the blood, and not "the harmless sugar," set up the diseased condition of the uveal tract, which in turn produces the cataract.

Several writers have cited cases of diabetes in which the lenticular opacities have disappeared when the general condition improved under treatment, proving that such opacities were not the result of complete degeneration of the lens fibres.

Before operating for the extraction of cataract in the case of a diabetic patient, it is well that some dietetic and constitutional treatment be inaugurated to improve his general condition. After observing this precaution and providing the condition of the other structures of the eye does not contraindicate it, the operator may proceed with reasonable assurance of success.

Limitation of the power of accommodation is a well-recognized symptom not infrequently met with in even mild cases of diabetes, and the early development of presbyopia should always lead to examination of the urine. This, as well as the occasional mydriasis, is attributed by some to the general muscular weakness, by others to a peripheral neuritis or to hemorrhages, while still others consider that it is due to toxic substances circulating in the blood.

Diabetic myopia has been reported from time to time, and while in some instances it is apparently due to other causes, in a large proportion of cases it is probably due to swelling of the crystalline lens in the early stages of a diabetic cataract. Myopia developing in patients past forty or fifty years of age should always suggest an examination of the urine.

One case of acquired hypermetropia was reported by Horner[1] in a patient fifty-five years of age who had severe diabetes. The hypermetropia diminished when the diabetic condition improved under treatment. This case seems to be unique, and is difficult to account for.

Retinitis, while seldom seen early in the course of diabetes, is of frequent occurrence in the terminal stages, presenting itself in an exudative or hemorrhagic form, or with exudations and hemorrhages combined. Appearing at a period in the course of the disease when degenerative changes in the vessel walls are present in other parts of the body, as well as in the eye, it is not strange that the glycosuric form is sometimes seen in association with albuminuric retinitis. In the exudative form there are apt to be small, light, shining patches with minute hemorrhages, but swelling of the retina and involvement of the nerve are not characteristic, as in albuminuria. Hemorrhages, either in the small punctate form or larger and of sufficient extent to lead to the formation of vitreous opacities, are frequently seen.

[1] Klinische Monatsblätter für Augenheilkunde, 1873, S. 490.

Several writers have reported cases of hemorrhagic glaucoma, and while small hemorrhages are seen frequently in cases which yield for a time to treatment, extensive retinal hemorrhages of diabetic origin are of the gravest prognostic significance.

Among the rarer results of diabetes we sometimes have choked disk, neuritis, and neuroretinitis, with secondary atrophy.

Amblyopia and amaurosis, which are referred to elsewhere, are also occasionally seen in the course of diabetes. With such amblyopia we may find central scotoma for red.

Diabetes Insipidus. As a result of or associated with diabetes insipidus, a number of writers have reported hemianopsia, epileptoid attacks, optic neuritis, and symptoms of cerebral tumor; but it is probable, as Knies has suggested, that in these cases the polyuria was only an incidental effect of a lesion in the floor of the fourth ventricle, which was the real cause of the symptoms referred to.

Hemorrhagic retinitis is said to have been found in diabetes insipidus.

Graves' or Basedow's Disease (Exophthalmic Goitre). Palpitation or irregular action of the heart, enlargement of the thyroid gland, and protrusion of the eyeballs are the characteristics of this disease, although in the early stages one or more of these symptoms may be absent. In well-marked cases the diagnosis will force itself upon the most unobserving, but it is in the early stages and atypical cases that its recognition is of the greatest value.

Actual protrusion of the eyeballs in advanced cases is a most marked symptom; but, as the writer has had occasion to verify by actual measurement in a number of instances in mild cases, this is often only apparent. Stellwag has described the persistent slight retraction of the upper lids, and von Graefe the lagging of the upper lid, which is often observed in looking downward. This latter, von Graefe's symptom, which may for a long time be the only symptom of the disease, was absent only twelve times in six hundred and thirteen cases investigated by Sharkey.[1] The retraction of the lids, which is so constant an effect of the instillation of cocaine, and to which Koller has called attention, suggests the early stages of Graves' disease, and apparently is due also to a direct stimulation of the sympathetic nerves supplying the orbital muscles.

The extreme protrusion of the eyeballs seen in advanced cases is attributed by some authorities to spasmodic contraction of the unstriped muscular fibres found in the orbit, but the majority of writers refer it to dilatation of the orbital arteries producing an undue development of the fatty and connective tissue. It is a significant fact, however, that the exophthalmos often diminishes after death.

The most serious consequences so far as the eye is concerned sometimes result from exposure of the cornea due to this protrusion of the eyeball, which may not be closed even during sleep, and these

[1] British Medical Journal, October 25, 1890.

seem to be the more serious the more rapidly the eye is pressed forward. Corneal ulcers, resulting in nebulæ or going on to sloughing, sometimes occur, and yield only when they are recognized early and treated with the greatest care by thoroughly suturing the lids, and thus affording protection.

Rarely diplopia and even marked paralysis of one or more of the external ocular muscles has been observed, and sometimes there is severe pain in the eyes, with profuse scalding lacrymation.

The pupils respond well to direct illumination and contract on convergence, although moderate dilatation and irregularity are sometimes observed.

Auscultation over the orbit sometimes will elicit a distinct vascular murmur, similar to the placental bruit.

Exophthalmos, while generally seen on both sides, is not very infrequently unilateral, and often varies in degree in the two eyes, and Knies calls attention to Hack's[1] interesting observation that the exophthalmos on one side has been known to disappear after the mucous membrane of the nose on that side had been cauterized; and Bolose[2] has obtained the same result. A few other similar cases have been reported, but, as Knies remarks, they are exceptional.

Iritis is not uncommon in severe cases, and epiphoria often is marked. Nystagmus and tremor of the eyelids are sometimes observed, and a degree of corneal insensibility is of frequent occurrence, which, as Knies has remarked, accounts for the infrequency of the winking movements in many cases of Graves' disease.

Atrophy has at times been observed in extreme cases as a result of stretching of the optic nerves, but ophthalmoscopic findings are rare, consisting of pulsation of the retinal veins and rarely of the arteries.

As severe cases of Graves' disease are apt to be accompanied by anæmia and neurasthenia, it is natural that with headache and vertigo we should often find all of the direct and reflex symptoms which accompany muscular and ciliary asthenopia due to other causes, especially if there is a coexisting error of refraction. In Graves' disease the visual fields are at times concentrically contracted, while the central vision and color sense may or may not be impaired.

Goitre, owing to pressure upon the veins of the neck, at times causes disturbance in the vascular structures of the eyes by venous stasis. The author has seen one case in which he was convinced of a causative relation existing between goitre and a central choroido-retinitis.

General glandular enlargement, or polyadenitis, has, at least in one instance reported by Konigstein, been accompanied by a number of small tumefactions along the optic nerve and the motor oculi, with extensive neuroretinitis and total unilateral ophthalmoplegia externa.

[1] Deutsche med. Woch., 1885, No. 25.

[2] Ann. d'Ocul., 1896, p. 260.

DISEASES OF THE DIGESTIVE SYSTEM.

Diseases of the intestinal tract and the associated organs are not infrequently the cause of diseases of the eyes, although undoubtedly their importance in this connection has at times been exaggerated. This is especially true of the disorders appearing during the period of dentition, to which all the ills of infancy are attributed so frequently.

During the period of eruption of the deciduous, and less frequently of the permanent, teeth, and later as a result of the various forms of caries and other diseases producing irritation of the terminal filaments of the fifth nerve, we may have most marked reflex symptoms of the eyes; but, as suggested above, in their zeal for finding a plausible explanation of obscure phenomena, both physicians and parents often seem to forget that there is such a thing as coincidence without the relation of cause and effect. Neurotic manifestations, such as nictitation, mydriasis, myosis, relaxation, and, more frequently, spasm of accommodation, and even disturbances of the external ocular muscles, such as insufficiency leading in some cases to diplopia, are undoubtedly relieved at times by the removal or treatment of a carious tooth. Whether these symptoms are due to a true reflex irritation, or the result of a lack of sufficient innervation during the existence of pain in the tooth, must be determined by a study of each case.

The writer was strongly impressed with the importance of this association by the result obtained in the case of a patient who, after general medicinal treatment extending over a period of many months, was completely relieved of the most distressing symptoms of dyspepsia accompanied by gaseous distention and insomnia, by the adjustment of a 2° prism base in on each eye, for the correction of insufficiency of the internal recti. This relief continued for almost a year, when, without other cause which he could discover, he found the symptoms all returning, and, as the most critical examination failed to reveal a change in either the refraction, presbyopia, or muscular balance, it seemed that we had exhausted our resources. The patient was a man of fifty years of age, an attorney in active practice, who spent his days in office work and his evenings in reading, and the failure to obtain relief was making him most unhappy, when a friend suggested to him that it would be well to give attention to a defective, "ulcerated" tooth which was causing him so little annoyance that my attention had not been called to it. This he did, and on the removal of the source of the dental irritation the gastric symptoms at once and permanently disappeared. This case illustrates most completely the general principle that dental disease and asthenopia resulting from muscular imbalance, two absolutely different conditions, but in both of which the terminal filaments of the fifth nerve are involved, may produce the same group of reflex nervous symptoms in a remote organ.

Iritis, keratitis, phlyctenulæ, and even glaucoma have been attributed to disease of the teeth; and it is undoubtedly the case that an

alveolar abscess, with or without involvement of the antrum of Highmore, may give rise to infectious processes in the orbit, lids, and eyeball, although such a relation is of rare occurrence.

Serious gastric and gastro-intestinal disease leads frequently to anæmia and other general diseases, which, in turn, cause most marked ocular symptoms; but these will be dealt with elsewhere. In all forms of gastro-intestinal disorders accompanied by violent vomiting the conjunctiva, retina, or choroid may be the seat of hemorrhage. This is a source of real danger, especially in those with defective vessel walls, after operations involving section of the eyeball, and should be a strong reason for the avoidance of general anæsthesia, where practicable, in such cases.

An irritable state of the mucous membrane of the digestive tract is seen often in children in association with phlyctenular conjunctivitis and keratitis, and the correction of errors of diet and regulation of the alimentary secretions are followed by such immediate improvement that one naturally is inclined to believe that there is some association between the disease of the eyes and that of the alimentary tract more intimate than would be indicated by the gradual improvement in the nutritive processes following such management and treatment.

Vascular engorgement due to constipation and the consequent straining at stool may, in those with weakened vessel walls, lead to conjunctival, orbital, retinal, or choroidal hemorrhages; and, according to Berger, prolonged constipation as well as chronic diarrhœa have been assigned as a cause of acute glaucoma. He also calls attention to the fact that prolonged diarrhœa, as in dysentery, may result in marked enfeeblement of accommodation, and that in the chronic diarrhœa of infancy the addition of an element of infection to the systemic depletion leads at times to keratomalacia.

In disease of the liver accompanied by jaundice, the discoloration of the conjunctiva is often noted before the skin is stained, and may remain after the skin has cleared.

Hemeralopia, subjective yellow vision, analogous to that of santonin-poisoning, torpor of the retina, and even keratitis, may also result from hepatic disease, and in acute yellow atrophy, retinal hemorrhages, due to septic substances in the blood, are not uncommon (Knies). Landolt believes that we may trace a more or less definite relation as existing between cirrhosis of the liver and pigmentary retinitis, as well as hemeralopia without pigmentation.

Scintillating scotoma and ocular migraine, which are considered elsewhere, are often associated with and apparently dependent upon torpor of the lower bowel, and other disorders of the digestive system.

The peculiar yellowish plates in the skin of the eyelids, known as xanthoma palpebrarum, are associated frequently with disease of the liver; and Foerster, Seegen, and Hlawatschek report cases of failure of accommodation and equatorial lenticular opacities due to the same cause. Intestinal parasites, as is well known, often cause dilatation of the pupils and other reflex ocular symptoms.

DISEASES OF THE RESPIRATORY TRACT.

Diseases of the nose and pharynx are not infrequently the cause of disease of the eyes, and unless this fact is borne in mind, treatment is often unavailing. In many cases a source of irritation in the nose will produce reflex symptoms in the eyes which, although they may not lead to organic disease, are still most distressing. In other instances well-defined organic lesions are produced, although this is not of as frequent occurrence as would be inferred from the amount of literature on the subject. In this, as in many other departments of medicine, there is a curious discrepancy between the opinions expressed by Continental and by American observers. The former are disposed to discredit many of the reported cures of eye disease by the removal of sources of reflex irritation by operation and treatment of the nose.

A spur on the septum or a septum so deflected as to have its convex surface in contact with the adjoining turbinated body, hypertrophy of the middle or even of the inferior turbinated body, or any other form of nasal disease or deformity which may be accompanied by hyperæsthesia of the mucous membrane of the nose, may produce reflex symptoms in the eyes. These may cause pain and photophobia, lacrymation, persistent or recurring conjunctival and ciliary injection, blepharospasm, accommodative or muscular asthenopia, limitation of the visual field, amblyopia, and in those who are predisposed, may even excite glaucoma. Cheatam and Lennox Browne have reported cases of glaucoma in which nasal irritation served as an exciting cause.

The intimate association existing between the nasopharyngeal cavities and the various structures within the orbit, including the eye, is well illustrated by the temporary concentric narrowing of the visual field which in a number of instances has been known to follow the use of the galvanocautery in treating diseases in the nasal cavities. Knies considers this as a form of traumatic hysteria, and mentions the fact that similar symptoms may result from other painful procedures in the neighborhood of the eye, but this explanation is scarcely consistent with the results reported by Hack[1] and Hopmann,[2] in which Graves' disease was cured by the application of the galvanocautery to the nasal mucous membrane.

Rhinitis in its various forms, especially those associated with active infective processes, is among the most frequent causes of disease of the conjunctiva and cornea, and, while the nasal duct is the usual channel of communication, it is by no means impossible for germs or their products to be conveyed from the nose to the eyes by other means.

That it is possible, on the other hand, for fluids containing infectious material to be communicated from the eye to the nose is well

[1] Deutsche med. Woch., 1886, No. 25.

[2] Jahr. f. Augenheilk., 1885, p. 473.

established. And, although, because of tumefaction of the mucous membrane in infectious diseases of the conjunctiva, the duct is often occluded, it is probable that such material not infrequently passes into the nasal cavity with the tears, there to be rendered less potent by the secretions from the nasal mucous membrane.

The ease with which fluids may reach the nose and nasopharynx should always be borne in mind when using mydriatics and other substances in the conjunctival sac; and it should also be remembered that solutions of atropine, as they must frequently be used in the treatment of iritis, contain in the amount instilled far more than the usual pharmacopœial dose of that remedy, and it is probable that a larger proportion is absorbed than is the case when administered by the mouth in therapeutic doses.

Transmission of disease through the nasal duct from the conjunctival sac to the nose is certainly extremely rare, but Knies mentions lupus and epithelioma as having been so transmitted.

In acute catarrhal conjunctivitis the nasal mucous membrane seldom becomes secondarily involved; but, on the other hand, the conjunctiva seldom escapes in an acute coryza, while in the chronic forms of rhinitis the lacrymal sac or conjunctiva is very prone to be affected at some period, especially during acute exacerbations and in the atrophic stage, when crusts form near the nasal orifice of the duct. Syphilitic coryza is seldom transmitted by this channel, but Knapp has described a case of lupus of the nasal cavity which produced tubercular conjunctivitis. In many of the infectious diseases which affect both the conjunctiva and the nasal mucous membrane there seems to exist no evidence that there has been transmission from one to the other. Even in the case of diphtheria and croup in which the nasal and, much more rarely, the conjunctival mucous membrane are involved, there seems to exist no proof that the path of the infection has been through the nasal duct.

Adenoid vegetations in the vault of the pharynx, and hypertrophy of the pharyngeal tonsils, are, especially in children, frequently found bearing a causative relation to diseases of the eyes. There seems to exist a somewhat intimate relation between these growths with their associated conditions, and phlyctenular conjunctivitis and keratitis.

Disease of the frontal, ethmoidal, and sphenoidal sinuses due to inflammation of their lining membrane or occlusion of their outlet, accompanied by the accumulation of serous, mucous, or purulent fluid, or the formation of granulation tissue or various forms of tumors, may all most profoundly affect the eyes, and for long periods the nature of the trouble may escape detection.

Supra-orbital or, more rarely, infra-orbital neuralgia, pressure symptoms, and reflex phenomena, displacement of the eyeballs with ptosis, and disturbed relations of the extrinsic ocular muscles, may occur; and in the early stages, before the development of marked protrusion, the writer has in two instances seen on ophthalmoscopic examination a peculiar parallel striation of the retina.

When erosion or marked inflammation of the bony walls of the orbit is present, we may have an orbital cellulitis or abscess accompanied by chemosis and interference with corneal nutrition, and consequent sloughing. If the sphenoidal sinus is involved to such a degree as to cause necrosis of its walls, we are apt to have first disturbance of the visual field, and later paralysis of the external ocular muscles, and blindness, from disease of the optic nerve, chiasm, or tract.

Diseases of the ear, which may properly be considered in connection with the superior respiratory tract, may in rare instances produce eye symptoms. As illustrations of reflex nervous phenomena may be mentioned blepharospasm, which sometimes occurs on irritation of the external auditory meatus, and the nystagmus which Pflüger observed on compressing a polypus.

Mastoid disease or operations for its relief may cause lagophthalmos by partial or complete paralysis of the facial nerve, although the operative form often recovers. When meningitis or cerebral abscess occurs, we may have the usual results in paralysis of the external ocular muscles, optic neuritis, etc.; and Kipp[1] and Pomeroy[2] have reported three cases of metastatic panophthalmitis in purulent otitis media.

Diseases of the larynx, trachea, and bronchial tubes are not often associated with disease of the eyes, although a number of curious reflex phenomena are often observed, such as dilatation of the left pupil accompanying infiltration of the apex of the left lung, and the sneezing produced by opening the eyes, especially in cases of conjunctivitis and keratitis.

Bronchitis and pneumonia may be accompanied by herpes of the cornea; and the dyspnœa of emphysema is, as Knies has pointed out, accompanied by stasis in the retinal veins, and at times conjunctival and retinal hemorrhages. Schmall has often seen injection of the fundus, and reports five cases of visible arterial pulsation in phthisis. Neuroretinitis has been reported in association with pneumonia; and Gowers describes a case of intense febrile bronchial catarrh with marked cyanosis, which was accompanied by neuroretinitis with enormous extravasations, many of which were regularly arranged and situated upon the smaller veins. The writer has, on the other hand, seen a fatal apparently metastatic pneumonia develop in the course of panophthalmitis following gonorrhœal ophthalmia.

DISEASES OF THE CIRCULATORY SYSTEM.

Diseases of the circulatory system manifest themselves in the eye by hyperæmia, anæmia, œdema, hemorrhage, and the results of hemorrhage in the conjunctiva, uveal tract, nerve, and retina; and when the blood is modified by disease or is the bearer of effete material

[1] American Journal of the Medical Sciences, April, 1884.
[2] New England Medical Monthly, January, 1889.

or toxins generated in other portions of the body, we may have also embolism, thrombosis, and various forms of exudate within the choroid and retina. The peculiar anatomical arrangement of the circulation within the eye should be borne in mind. Its supply of blood is derived from both the internal and external carotid arteries. The free anastomosis of these two systems and the communication with the opposite side of the brain are such as to provide well for its nutrition, even when serious obstruction exists in some of the large vessels. Especial attention should be directed to the large, freely anastomosing arterial and venous trunks of the tunica vasculosa of the choroid, overlaid by the chorio-capillaris, which affords nutrition to the layer of rods and cones, or epithelial layer, while the terminal system made up of the central retinal artery and vein with their branches supplies the inner or "brain layer" of the retina.

While there has been in the past a tendency to expect too much of the ophthalmoscopic appearance of the vascular system of the eye in interpreting the phenomena of the general vascular system, and especially the conditions existing within the cranial cavity, and while its limitations as an index of these conditions should constantly be borne in mind, the fact remains that when properly interpreted they are of the utmost value.

In estimating the value of ocular manifestations, a sharp distinction should be drawn between those conditions observed in the retina which are associated with evidences of disease of the blood itself, with the accompanying degeneration of the vessel walls, and those due merely to alterations in blood volume and blood pressure. The peculiar conditions of the circulation within the eyeball, modified as they are by the non-elastic sclerotic coat, and by the admirable arrangement by which under ordinary circumstances the large trunks of the choroid serve to regulate the pressure upon the visible retinal circulation,[1] renders it impracticable, as has been attempted, to use the retinal circulation as a means of gauging the blood pressure in the vascular system generally, or even in that of the brain.

General anæmia may be accompanied by more or less hyperæmia of the eyes, and it is only in extreme cases that variations of blood pressure, so easily recognized in other portions of the vascular system, can be properly interpreted by means of the ophthalmoscope.

Hyperæmia of the conjunctiva may be present as a local manifestation where there is a corresponding condition of the general circulation due to various diseases, but it is not a very consistent sign, as many cases of most obstinate conjunctival hyperæmia accompany general anæmia. Hyperæmia of the retina and optic nerve is not apt to be found as the result of general plethora, but is more frequently of the passive form, and accompanies such obstructive conditions as asthma and emphysema, and such heart lesions as induce venous stasis.

[1] It is probably to a disturbance of this delicate balance that many of the phenomena of glaucoma are owing.

The eye is far better able to protect itself against the ill effects of a sudden increase of blood pressure than against sudden diminution of pressure, although it is an extremely rare occurrence to have retinal hemorrhage or other permanent retinal disease as a result of either, unless there is some disease of the walls of the bloodvessels.

The acute anæmia due to extensive hemorrhage is only in rare instances followed by disease of the eyes. Fries[1] was able to find the record of only 106 cases that have occurred during the past two hundred and thirty-five years; and of these, it is probable that many occurred among those having some form of disease of the bloodvessels. Sixty per cent. were from gastric, intestinal, and uterine hemorrhage, 25 per cent. from artificial abstraction of blood, 7 per cent. from epistaxis, 5 per cent. from wounds, and only 1 per cent. from pulmonic hemorrhage.

That serious disease of the eyes results only in the rarest instances from extensive hemorrhage in individuals with healthy bloodvessels is apparent when we recall the enormous number of instances of profuse hemorrhage occurring as the result of traumatism, especially during military engagements, and note the extreme infrequency of disease of the eyes as a result. Not a single case is recorded as having occurred during the Franco-Prussian War of 1870–71. When amaurosis and amblyopia do occur as the result of extensive loss of blood, it is usually after several days have intervened, and it seems to be due to hemorrhage into the optic nerve, resulting from fatty degeneration of the vessel walls consequent upon disturbance of nutrition from insufficient supply of blood (Knies).

From the above statistics it is apparent that extensive hemorrhage from the stomach, bowels, and uterus is a real source of danger to vision; and, having in mind this danger and the weakened state of the bloodvessels, the physician will naturally insist upon the recumbent posture, and will practise intravenous injections or adopt such other measures as may tend to restore the equilibrium of the vascular system.

In aortic insufficiency the rhythmical reddening and bleaching sometimes observed in the finger-nails may be apparent in the optic disk (Jaeger), and pulsation of the arteries in harmony with the radial pulse is also generally seen (Haab).

Other cardiac diseases occasionally affecting the eye are mitral insufficiency, dilatation of the heart, and fatty degeneration. The latter disease is often only one manifestation of a general process, of which one of the features is disease of the ocular vessels.

Among the diseases of the vascular system which sometimes, although rarely, affect the eyes may be mentioned aneurism of the aorta, arteriovenous aneurism of the internal carotid and the cavernous sinus, producing a pulsating exophthalmos, and, very rarely, aneurism of the ophthalmic artery.

[1] Klin. Monats. f. Augenheilk., 1876.

In aneurism of the aorta we may have paralysis of the sympathetic nerve, which leads to contraction of the pupil and also of the palpebral fissure of the affected side.

A local, temporary slowing of the blood current, due to traumatism or other cause, producing a thrombus from which small masses may break away, foreign substances in the bloodvessels, and endarteritis in its various forms, causing fibrinous deposits on the vessel walls or the valves of the heart, may result in the development of embolism of the central artery of the retina or one of its branches. In some sixteen cases which have been studied anatomically the obstruction was, in most instances, found in the region of the lamina cribrosa (Haab).

In those portions of the general vascular system in which there is free anastomosis, such emboli, even if they find lodgement, may cause only very slight and temporary disturbance, and this is in large measure true of the vessels of the choroid; but in the retina. as in many portions of the cerebral cortex, we have one of the terminal systems of Cohnheim, in which more or less complete cutting off of nutrition occurs in the area supplied by the obstructed vessel.

Thrombosis of the retinal vessels may occur as a complication of inflammatory and especially of infectious processes in the orbit, whether they are caused by erysipelas, meningitis, thrombosis of the cerebral sinuses, or in any other manner (Haab). They may involve both the arteries and veins, and are apt to be the immediate effect of stooping or straining in people having disease of the bloodvessels.

Unless the patient is seen soon after the accident has occurred and the case is studied with great care, it is in many instances by no means easy to make a clear distinction between embolism, thrombosis, and endarteritis obliterans. The presence of perivascular infiltration and degeneration accompanying endarteritis, and visible to the ophthalmoscope, is indicative of thrombosis, and an existing valvular lesion of the heart is characteristic of embolism.

In some cases, however, as in several which have been observed by the writer, the sudden occurrence of the symptoms and the ophthalmoscopic picture are such as to leave no room for doubt as to the existence of an embolism.

DISEASES OF THE URINARY ORGANS.

Albuminuria. Among the diseases usually grouped under this head, albuminuria (Bright's disease) in its various forms is by far the most important, and the one in which eye lesions are found most frequently. Œdema of the integument of the eyelids of a temporary character may occur at a very early period in nephritis; but permanent œdema, when present, usually accompanies œdema in the ankles and other parts of the body as a late manifestation.

Chemosis or œdema of the conjunctiva is rare.

Among the intra-ocular affections due to albuminuria we have hyperæmia of the papilla and retina, retinitis with the characteristic white spots arranged in radiating lines about the macula, neuritis, neuroretinitis, and even choked disk with hemorrhages, especially, in the nerve-fibre layer.

Detachment of the retina, iritis, and hemorrhagic glaucoma have been described as rare conditions, and choroiditis as of not infrequent occurrence. Changes due to the latter disease have often been found on autopsy; but the lesions, which seldom are reported, are probably rendered invisible at the ophthalmoscopic examination by the pigment epithelium (Knies). Whitish patches, generally in the macular region, but not infrequently in other portions of the central retina, accompanied by swelling of the nerve-fibre layer and, at times, by small, often flame-shaped hemorrhages, are the characteristic retinal manifestations of albuminuria on ophthalmoscopic examination.

Extensive hemorrhage into the vitreous is seen sometimes, but it is of rare occurrence.

Complete blindness is very rare, seldom occurring, excepting in coincident uræmic amaurosis or in atrophy of the optic nerve and detachment of the retina. Retinitis is not an early symptom, but it is not infrequently the first symptom which leads to a correct diagnosis. It has occasionally been the experience of the writer, in patients having almost normal vision and using their eyes quite constantly, to find on making an ophthalmoscopic examination that the central fundus was the seat of extensive retinal infiltration. In these cases the fovea has escaped, and the conducting power of the axis-cylinders seemed to be unimpaired. The œdema may be marked, and yet the rods and cones may for a long time remain undisturbed.

The disease with which we are dealing affects the bloodvessels mainly as a sclerosis; and Knies has pointed out that all the other lesions, including the hemorrhages, œdema, the formation of folds, and detachment of the retina, and fatty degeneration, are secondary to these changes in the bloodvessels. He calls attention also to the fact that while disease of the choroidal vessels may be very extensive without giving rise to such nutritive disturbances as are seen in the retina, it is because in the choroid we have a collateral supply, while in the retina the arteries are end arteries, and circulatory disturbances are not compensated.

There is marked thickening, especially of the intima, of the small arteries and capillaries; and while in the latter we often find dilatation, in the former small dissecting aneurisms are not uncommon.

Weeks reports a case of hemorrhage and acute glaucoma occurring with albuminuric retinitis, and other cases have been recorded proving that acute glaucoma is one of the occasional complications of this disease. Bull has pointed out that disease of the walls of the bloodvessels is probably the connecting link between these two conditions; and Gowers states that in some cases of chronic renal disease there

is diminution in size of the retinal arteries independently of the existence of other evidence of special renal disease.

Albuminuric disease of the retina is to be found in all forms of nephritis; but it is rare in the large white kidney of parenchymatous nephritis, in the stage of fatty degeneration, and also in the form characterized as waxy kidney. In this latter form Bull has pointed out that it is seen only when the waxy degeneration occurs in a contracted kidney. Although not uncommon in the albuminuria of pregnancy, and sometimes seen in the acute forms of nephritis, such as appear as a complication of scarlatina, all observers seem to agree that changes in the retina are found most frequently in the late stages of that form of nephritis which is accompanied by contracted kidneys. They make their appearance when, after a period of high vascular tension, elimination is beginning to fail, but are by no means always found under these conditions.

While perhaps somewhat less serious, if the nephritis is of the form which is often seen in acute exanthematous diseases and in pregnancy, the prognosis is always grave, or at least doubtful, the patients often dying within a few months after the discovery of the retinitis.

Knies, in speaking of the more chronic forms of nephritis, states that life is rarely prolonged more than one, or at most two years after the discovery of retinal infiltration; but when seen among those who will carefully observe the hygienic and other regulations necessary under such circumstances, it has not, in the writer's experience, been uncommon to see that period greatly extended. In the albuminuria of pregnancy, threatened loss of sight from neuroretinitis is a strong indication for the induction of premature labor.

Though generally affecting both eyes, unilateral albuminuria is observed occasionally. Bull has described ten cases.

Lesions of the retina which resemble those of true albuminuric retinitis, undoubtedly occur as a result of profound anæmia, leukæmia, and diabetes, and in some forms of organic disease of the brain, as well as in a certain proportion of cases of what is termed cyclical albuminuria; but it is well in these cases to make frequent tests of the urine, for, as is well known, nephritis may coexist with these conditions, and albumin be absent from the urine for a considerable period. While rare, paralyses of the external ocular muscles are seen sometimes in this disease, occurring as the result of a hemorrhage in the region of the nerve roots or nuclei (Knies).

With uræmic amblyopia or amaurosis, muscular paralysis may at times be seen in the last stages. These are not necessarily accompanied by changes in the retina. When uræmic poisoning occurs in acute nephritis, as in that of pregnancy, it is not uncommon to find that the retina has undergone no change whatever, and, in fact, it has been pointed out that the combination of uræmic amblyopia and retinal disease is comparatively rare. In eclampsia the pupils are generally dilated, and the external ocular muscles often take part in

the convulsions. Cataract may be the result of vascular changes dependent upon nephritis.

Oxaluria, uric acid diathesis, and **phosphaturia** have each in rare instances been assigned as the cause of eye lesions, but it may well be doubted whether the changes observed in the eyes were properly to be attributed to these conditions or to some other unrecognized disease on which they in turn were dependent.

Hansell[1] recorded a case in which the retinal changes, although not quite characteristic, resembled those of albuminuric retinitis. There was failure of vision, but there were no subjective symptoms. The abnormally abundant urine contained an excess of phosphates, but repeated examination revealed no albumin or sugar.

DISEASES OF THE SEXUAL ORGANS.

Much has been written on the effects of sexual excesses, especially masturbation, in producing disease of the eyes, and, if we were to be influenced by the extreme views of many otherwise high authorities, we would conclude that there exists some peculiarly intimate relation between the eyes and the sexual organs. If such a relation exists, its importance has certainly been vastly overestimated.

Sexual excesses have been assigned as a cause of atrophy of the optic nerve, but it is doubtful if the relation of cause and effect has been established.

Conjunctival hyperæmia, catarrhal inflammation, impairment of accommodation, muscular asthenopia, and even serious disease of the optic nerve, have all been attributed to habitual masturbation, both in male and female patients.

It is undoubtedly the case that a degree of neurasthenia often results from the direct and indirect effects of this morbid habit, which in its turn serves to aggravate asthenopic conditions due to other causes. In common with other conditions leading to vascular engorgement, excessive masturbation as well as venereal excesses have been known in numbers of instances to serve as the exciting cause of various forms of intra-ocular and subconjunctival hemorrhages when the vessel walls have been weakened by disease.

Gonorrhœa, which might for some reasons be classed more properly with the infectious diseases, is treated here because it is so essentially a disease of the genital mucous membrane. Ophthalmia neonatorum and gonorrhœal ophthalmia in the adult, with the resulting destructive keratitis, are the well-known eye complications of this disease.

Metastatic disease of the eye is also sometimes seen in association with gonorrhœa, especially with gonorrhœal rheumatism of the larger joints, and iritis not infrequently accompanies the rheumatism due to this disease. Well-authenticated cases of metastatic gonorrhœal

[1] Philadelphia Polyclinic, January 21, 1897.

conjunctivitis, with intense chemosis and scanty non-purulent secretion, but without destructive corneal complications, have been reported.

To the various forms of disturbance in the vascular and nervous systems which are excited by abnormal menstruation are properly attributed a large group of ocular affections, and in a still larger number of instances diseases of the eyes due to other causes are aggravated by such disorders. Patients who are anæmic, chlorotic, or "scrofulous," especially, and sometimes those free from such conditions, show a marked tendency to disease of the eyes during or immediately prior to menstruation. This may be insignificant, and manifest itself as a slight œdema of the lids, or by the appearance of dark rings under the eyes, or it may be that conjunctival hyperæmia will develop or well-defined asthenopic symptoms present themselves.

Given a tendency to herpetic eruptions of the lids or cornea, to marginal blepharitis, styes, phlyctenular conjunctivitis or keratitis, or even iritis, the approach of the menstrual period, especially if there be dysmenorrhœa, is apt to be accompanied by an outbreak.

In nervously susceptible patients a variety of symptoms of an hysterical character may present themselves at the menstrual period, such as limitation of the visual fields or modification of the color fields.

Leber[1] calls attention to the possibility of hemorrhages into the optic nerve during menstruation in cases in which the vessels are diseased, and Knies mentions cases of hemorrhages into the conjunctiva, vitreous, and anterior chamber.

Attacks of anterior uveitis and disseminated choroiditis and choroidoretinitis are frequently due to menstrual disorders, and sudden suppression of the menses is said, in some instances, to have produced hemorrhages into the optic nerve and its sheath.

Hemorrhagic glaucoma is sometimes seen at the time of the menopause.

It is doubtful whether normal pregnancy and normal parturition in a healthy woman ever are accompanied by diseases of the eyes; but, as in menstruation, a pre-existing disease of the eyes, a systemic condition, such as anæmia, which seriously interferes with the normal course of pregnancy, or any of the numerous accidents which may befall the patient during this critical period, may convert what should be a normal physiological process into a prolific source of disease of the eyes.

In a nervously susceptible woman, especially if she is anæmic or is the subject of any form of disease which impairs her vitality, muscular or accommodative asthenopia may become a source of great discomfort, and if, as we frequently find, there exists a latent error of refraction or imbalance of the external ocular muscles, the symptoms

[1] Handb. v. Graefe-Saemisch, Bd. v. p. 819.

are aggravated and are sometimes most distressing. In these cases as in those of an hysterical nature, such as temporary blindness unaccompanied by organic lesion, and contraction of the visual fields, we should attribute the disturbance to the anæmia or other systemic condition upon which it really depends, and look upon the pregnant state as merely an exciting cause.

The eyes bear their part in the altered facial expression sometimes seen during pregnancy, and are not infrequently the seat of pigmentation. Phlyctenular conjunctivitis and even keratitis sometimes are encountered.

The occurrence of albuminuria during the later months of pregnancy is apt to be accompanied by retinitis, choroiditis, optic neuritis, etc., which are considered in their respective chapters. In properly selected cases the induction of premature labor may check the progress of the disease, and, while not infallible, has in many instances served to prevent blindness. As, however, the same accident is apt to occur in future pregnancies, patients should be warned of their danger. Detachment of the retina and retinal hemorrhage may occur even when not associated with albuminuria.

It is natural that parturition, especially when painful or prolonged, should in cases in which albuminuria or other disease has weakened the walls of the vessels, lead to hemorrhage into the retina and nerve.

Amaurosis may make its appearance during parturition as a result of the uræmia of eclampsia, or when there has been profuse uterine hemorrhage it may result, as in cases of great loss of blood from other portions of the body.

Hemorrhages into the retina and optic nerve occurring during childbed without apparent cause are attributed by Knies to emboli of the central retinal artery, such as have been observed after phlegmasia alba dolens.

Puerperal septicæmia may lead to metastases in the retina and choroid, and in severe cases septic embolism may readily be followed by panophthalmitis. It is probable that, as in other forms of septicæmia and pyæmia, this occurs far more frequently than is reported, as attention is directed to other symptoms and the patients so seldom recover.

Abortion accompanied by infection, and septic processes in the womb occurring as the result of disease, or following the various surgical procedures involving the womb, may, in a similar manner, lead to disease of the choroid, retina, and optic nerve.

To lactation and the anæmia and exhaustion which at times accompany it are frequently due phlyctenular and other forms of keratitis, and even choroiditis accompanied by vitreous opacities has been found to be due to this cause.

The eyes of infants are often injured at the time of birth. This may occur in a variety of ways in prolonged but otherwise normal labors, but is especially apt to occur in cases in which instrumental delivery becomes necessary. Here almost every form of traumatism

has been recorded. Ecchymosis into the conjunctiva, hemorrhage within the eye or orbit, and fracture of the frontal bone or at the base of the skull, with resulting neuritis, followed by paralysis of the optic nerve, may be encountered, and we may have paralysis of any branch of the third nerve, the sixth, or the facial. Numerous cases in which paralyses have occurred have been published by Budin, Bloch, Berger, and others. But when we consider the numerous instances in which instrumental delivery is resorted to, serious accidents to the eyes are seen to be of relatively infrequent occurrence.

Ophthalmoscopic examinations of the newborn have in many instances revealed retinal hemorrhages, and while such eyes often recover with good vision, there is much reason to believe that many of the cases of amblyopia so frequently seen in strabismus are due to such lesions, the gross changes, such as could be seen by means of the ophthalmoscope, having long since disappeared. There is room for further investigation of this subject.

Ophthalmia neonatorum, almost always due to the gonococcus of Neisser, but sometimes traceable to other sources of infection, is so common as to require little comment. As in gonorrhœa, it may be associated with affections of the joints.

POISONS AND INFECTIOUS DISEASES.

Poisons. The direct and indirect effects of the introduction into the system of chemical poisons and living germs and their morbific products are so varied that it is extremely difficult to arrange a system of classification which will be thoroughly scientific and satisfy all of the conditions. Indeed, for our present purpose no such classification is necessary, as our object will be attained if, by grouping similar forms of poisons on the one hand and the infectious processes on the other, we are able to present a comprehensive view of the manner in which the eye is affected directly by the substances belonging to the various groups, or indirectly by the diseased conditions which they excite in other parts of the body.

A large and most important group of poisonous substances affect the eyes by producing some form of retrobulbar neuritis, at times including a lesion of the ganglion cells, resulting in what is generally termed toxic amblyopia, characterized by form and color scotomata. To this group belong alcohol, tobacco, lead, arsenic, the silver salts, mercury, phosphorus, the salts of potassium, iodoform, ioduret and thiuret, essence of Jamaica ginger and essence of peppermint, bisulphide of carbon and chloride of sulphur, nitrobenzol and dinitrobenzol, the various coal-tar products, opium and its alkaloids, chloral, cannabis indica, tea, coffee and chocolate, ergot, vanilla, and stramonium, carbon dioxide, osmic acid, quinine and the various products of cinchona bark, salicylic acid, and aspidium or filix mas. Each of these substances, it has been claimed, has produced true toxic am-

blyopia "by an action on the ganglion cells, nerve fibres, and optic nerves, or their vascular supply" (de Schweinitz), and while their general effect upon the eyes will be considered here, the symptomatology and pathology of retrobulbar neuritis will be treated of in the chapter on Diseases of the Optic Nerve.

As the term poison is applied to those substances which, if introduced into the system, produce disease, we here consider not the effect of all chemical substances upon the eye, but such effects as are produced in that organ when a given poison is introduced into the system or directly into the eye, and we should make a distinction between those effects which follow an acute attack and those which gradually make their appearance in cases of chronic poisoning. In both acute and chronic poisoning, however, the more pronounced eye symptoms are often the indirect effect of the action of the poison upon some other part of the body, such as the vascular system, the central or peripheral nervous system, or the kidneys.

The indirect effects upon the eye of acute systemic poisoning are apt to manifest themselves in the action of the iris and ciliary body, as mydriasis, myosis, cycloplegia, etc., and sometimes in the external ocular muscles and in changes in the vessels of the fundus. Colored vision and illusions are also sometimes noted.

While intoxication amblyopia is the most noteworthy effect of the substances named in the above list, a number of them produce other eye symptoms of which we must not lose sight.

Chronic lead-poisoning produces a variety of both central and peripheral lesions, resulting in disturbances of vision which are generally traceable to sclerosis and periarteritis, the evidence of which may sometimes be seen with the ophthalmoscope. Disturbances of the external ocular muscles may follow multiple neuritis due to this cause, and we may have the characteristic picture of albuminuric neuroretinitis, as albuminuria may result from lead-poisoning. The application of lead lotions is frequently followed by indelible deposits of lead in the cornea.

Phosphorus-poisoning is sometimes accompanied by hemorrhages in the retina, and later by fatty degeneration, somewhat resembling that of albuminuric retinitis. The frequent occurrence of hemorrhages in the optic nerve and brain produces characteristic eye symptoms.

Acute mercury-poisoning seldom produces eye symptoms, although in severe cases retinal hemorrhages and fatty degeneration, as in phosphorus-poisoning, have been noted. The eye symptoms in chronic cases resemble those due to chronic lead-poisoning.

Poisoning from the silver salts, aside from rare cases of retrobulbar neuritis, manifests itself in the eye only in the dark indelible stain of the conjunctiva, which not infrequently is seen as the result of prolonged local applications.

Iodine-poisoning, chiefly when it is administered in the form of iodide of potassium, induces at times catarrhal conjunctivitis, but

more frequently pains in the eyes and lacrymation in association with the typical coryza. We sometimes see illustrations of the extreme irritation produced by the development of iodide of mercury when levigated calomel is dusted into the eyes while iodide of potassium is being administered internally; but, although these drugs are not infrequently used at the same time, this is an extremely rare occurrence.

Bromide of potassium in excessive amounts has been known in some instances to produce retrobulbar neuritis, but even when not in sufficient dose to cause the characteristic skin eruption, it may excite conjunctivitis with phlyctenular foci of inflammation (Knies).

Alcohol, which probably produces more cases of chronic poisoning than all other substances together, causes a variety of eye symptoms in addition to retrobulbar neuritis, which latter disease will be considered elsewhere.

In acute alcoholism we have at times as an early manifestation, failure of accommodation, inco-ordination of the ocular muscles, causing diplopia, and later, absence of normal pupillary reaction. With normal ophthalmoscopic appearances we may, in delirium tremens, have a well-marked concentric limitation of the visual field which sometimes continues for several days. What is known as retrobulbar or axial optic neuritis is only one manifestation of the interstitial and parenchymatous changes and vascular disease due to chronic alcoholism, and the consequent lesions which develop in the brain, spinal cord, kidneys, etc., are accompanied by such changes in the eyes as are found in association with the same lesions due to other causes.

Severe disease of the central nervous system, such as often supervenes in cases of chronic alcoholism, is accompanied by a variety of eye lesions, and among them, in rare instances, ophthalmoplegia externa, which is due generally to hemorrhagic inflammation of the floor of the fourth ventricle.

Methyl alcohol and *amyl alcohol* may both cause sudden blindness—indeed, some authors have claimed that to these substances, rather than to pure ethyl hydrate, is due the retrobulbar neuritis which often follows the use of the more common forms of alcoholic drinks.

Acute opium-poisoning causes well-marked myosis and, at times, clouding of vision. In chronic cases accompanied by marasmus there is also apt to be corneal softening.

Acute chloral-poisoning produces myosis, although after large doses have been administered for a long time mydriasis sometimes is noted. The cutaneous eruption and urticaria of the lids, which are seen after prolonged use of the drug, are apt to be accompanied by conjunctivitis.

Sulphonal-poisoning may produce ptosis, and has been known to cause anæsthesia of the conjunctiva.

Carbon dioxide-poisoning has in a number of cases been known to produce paralysis of the external ocular muscles, which may disappear after a few weeks. It is attributed to hemorrhagic processes in the

nerve nuclei or in the peripheral nerves (Knapp). When obstruction to respiration or circulation results in the accumulation of carbon dioxide in the blood, we are apt to have retinal hemorrhages.

Santonin-poisoning is characterized by yellow vision, which, according to Knies, who has carefully investigated its phenomena, begins ten or fifteen minutes after the administration of the drug, and is preceded by transient violet vision. The pupillary reaction is normal, as is the appearance of the fundus, and central vision remains undisturbed.

Ergot has been known to produce narrowing of the retinal blood-vessels and temporary disturbance of vision, with sluggish pupillary reaction; but in chronic poisoning the prolonged nutritive disturbances occurring as the result of spasm of the vessels of the ciliary body are usually assigned as the cause of the cataract which sometimes follows within a few years of such an attack.

Fungus-poisoning varies in its effect upon the eye according to the nature of the alkaloid produced: muscarine causing spasm of accommodation and myosis, while other varieties, such as morchella, produce mydriasis (Knies). The profound systemic disturbance produced by these deadly alkaloids sometimes results in fatty degeneration and hemorrhage of the retina.

Atropine may be taken as the type of the mydriatics, and its effects upon the eye when used internally in a poisonous dose are well understood. It may cause hyperæmia of the fundus as well as cycloplegia and mydriasis, and in those who are predisposed may excite glaucoma. Not infrequently we find individuals in whom the smallest amount of atropine acts as a local poison in the conjunctival sac, producing a peculiar form of conjunctival catarrh and a so-called eczema of the lids. It is sometimes stated that this is due to impure solutions; but by rigid sterilization and the observance of every reasonable precaution the writer has convinced himself that this is not the case. Hyoscyamine or duboisine will usually be tolerated by those patients who are susceptible to this action of atropine.

Hyoscyamine, duboisine, daturine, homatropine, scopolamine, etc., have mydriatic and cycloplegic effects similar to those of atropine, and any of them may, under favorable conditions, induce glaucoma. Duboisine is more prone to cause delirium, and also, at times, produces narrowing of the visual field; and Pooley has noted temporary amblyopia as a result of the instillation of scopolamine.

Cocaine-poisoning if acute, may cause transient amblyopia with contraction of the retinal arteries, or it may produce syncope with the attendant temporary blindness. In chronic cocaine-poisoning we may have visual hallucinations, chromatopsia, hemianopsia, micropsia, diplopia, dancing of objects, and amblyopia. In a number of instances glaucomatous symptoms have developed after the instillation of cocaine into the conjunctival sac or after its use on the nasal mucous membrane.

Eserine instilled into the conjunctival sac has, in a few instances, produced temporary complete blindness and a degree

of transient amblyopia sometimes follows the injection of pilocarpine, but it is a curious fact that when administered internally both physostigmine and pilocarpine often produce mydriasis.

Landesburg has reported five cases in which opacity of the crystalline lens followed treatment with jaborandi.

Ptomaines and *toxalbumins* found in certain animals, or as the result of the action of microbes upon certain articles of food, such as meat, sausage, oysters, fish, ice-cream, etc., produce a variety of diseases of the eyes. Many of the ptomaines in their physical properties resemble certain of the vegetable alkaloids, which accounts for the fact that bilateral paralysis or paresis of accommodation and mydriasis are often observed after poisoning from decomposed meats or fish. Muscarine and neurine produce spasm of accommodation, while tyrotoxicon causes paralysis. Ptosis and paralysis of the extrinsic ocular muscles have also been observed, and have been considered as of nuclear origin, or possibly due to a basilar neuritis.

Infectious Diseases. Infectious diseases, their complications and sequelæ, are the cause of numerous eye lesions. Such diseases may be due to single micro-organisms producing well-defined clinical manifestations, such as are seen in glanders, tetanus, and splenic fever, and are in each instance peculiar to the specific agency which produces them, or they may result from the more complicated processes by which different microbes may be the cause of the same group of clinical symptoms, as is the case in erysipelas and pyæmia. In either instance the resulting eye lesions may be a direct effect of the primary infection, or an indirect result of the numerous complicating conditions which arise in the course of the disease.

The involved and intricate nature of the subject under discussion will be appreciated when it is remembered that with many of the infectious processes which are supposed to have a specific cause we may have in various stages of the disease complications arising from secondary infections. This is especially true of those accompanied by abscesses and purulent processes, the mixed infection being due to the addition of one or more of the common forms, such as Staphylococcus pyogenes aureus and albus, Streptococcus pyogenes, Diplococcus pneumoniæ, etc. Indeed, the clinical picture which is made up of a group of signs and symptoms to which we give a special name, is in many instances dependent for its existence upon the combined action of two or more of these micro-organisms and their morbific products. It is often to these secondary infections, which are so common that we look upon them as a necessary part of the disease, that are due many of the complications which make their appearance in the eyes.

Knies has made the following summary of the affections of the eyes which may occur in connection with infectious diseases:

1. Hemorrhages in all parts of the peripheral and central visual apparatus, from the most varied causes in all stages of the disease, and consequently every possible disorder of vision, motion, and sensation.

2. Foci of fatty degeneration and softening in the central organs and the eye, visible in the retina with the ophthalmoscope, and often associated with hemorrhages.

3. Inflammatory changes in the vessels in all localities, with the above-mentioned consequences.

4. More or less diffuse inflammations of the tissues of the eye, especially of the uvea and retina, iritis, cyclitis, choroiditis, retinitis, diffuse interstitial keratitis, etc. Meningitis with its various eye symptoms also develops in the same way.

5. Changes (chronic and acute hemorrhagic forms) in the optic nerve, chiasm, tractus, motor and sensory nerves (multiple neuritis).

6. Pure atrophy of the nerve tissues (central organs and optic nerve), occurring after the lapse of years, and probably the final outcome of the vessel lesions.

7. Focal hyperæmias and inflammations (metastases) in various degrees, from a chronic to an acute hemorrhagic and purulent process, or even terminating in acute gangrene. These are found in the integument of the lids (eruptions sometimes leading to gangrene), the sclera (sclerotic foci), uvea (disseminated choroiditis and choroido-retinitis, and embolic suppurations), retina (benign, but usually septic emboli), orbit (metastatic suppurations), lacrymal glands (embolic abscesses and dacryoadenitis), optic nerve, and brain, etc.

8. Specific neoplasms (syphilis, tubercle, leprosy) in almost every part of the eye and surrounding structures, and in the central nervous system.

The functional results of these lesions are:

1. Visual disorders of all kinds, of peripheral, intermediate, and central origin.

2. Paralysis and spasms of a central, nuclear, and peripheral character, and even due to direct disease of the muscles.

3. Neuralgias, anæsthesias, and paræsthesias of every possible mode of origin.

4. Other affections, such as adhesions of the lids in conjunctival catarrh, disorders of lacrymal secretion and conduction in affections of the lacrymal glands and canal, etc. In infectious diseases which are attended with high fever and congestion of the meninges and cortex, visual hallucinations and illusions also are encountered. These diseases may terminate in more or less severe forms of insanity.

The eye is in rare instances the seat of the primary infection, and not infrequently presents the earliest manifestations of disease which attract the attention of the physician or attendant, as in the photophobia and conjunctivitis often seen during the period of incubation of measles; but more generally eye lesions, when found in association with infectious diseases, are a direct effect of the disease itself or of some of its complications or sequelæ.

In studying the great variety of eye lesions produced by infectious diseases, it is well to bear in mind the marked difference which is often to be noted between those following an acute disease and those result-

ing from the more chronic processes. An infection may be so intense that, as in acute ptomaine-poisoning, the system is overwhelmed, and we find at the autopsy, in association with the other lesions, extensive retinal hemorrhage as the only ocular manifestation of a disease which, had its onset been less rapid, would have resulted in fatty degeneration in the parenchyma of the liver, kidneys and other organs, and the associated changes in the vascular system. These in turn would, in the more gradual process of infection, be accompanied by fatty degeneration in the choroid and retina and secondary hemorrhages in these structures similar to those found in the brain and other organs.

The profound infections which we often observe in such diseases as typhoid, typhus, and yellow fever, and in intense malarial poisoning, are accompanied by alterations in the blood, and at times by changes in the choroid, retina, and optic nerve, the exact nature of which cannot be said to be entirely understood, although they seem in many instances to bear a somewhat direct relation to the special poison causing the disease with which they are associated. The conditions attending the late war with Spain afforded many illustrations of the effects of these diseases in causing changes in the fundus of the eye, and a number of observers have made valuable contributions to our information on this interesting subject.[1]

It remains to be demonstrated, however, whether the lesions noted can be classed as part of the morbid anatomy of the diseases mentioned, or should be ascribed to intermediate changes dependent upon them, and possibly common to other maladies.

In the more chronic processes of infection in which the interstitial connective tissues are largely affected, or the vessel walls have undergone gradual degenerative changes, we have thrombosis and retinal hemorrhages, and as later manifestations, sometimes making their appearance years after the original lesion, atrophy of the optic nerve and retina associated with evidences of similar sclerotic changes in the brain and spinal cord.

As the general powers of resistance of the patient who is subjected to an infective process greatly modify the systemic effects produced, so the condition of the eye may influence the course of the disease in that organ. An old iritis with a posterior synechia, or an old penetrating wound with entangled iris or lens capsule, may make the eye far more vulnerable than it otherwise would be, and lead to most serious consequences when it is subjected to the dangers of an infectious disease.

A subject of great interest in connection with infectious diseases in their relation to the eyes is that which concerns the route by which the eye is invaded by the micro-organisms or their morbific products. In such diseases as lupus of the skin of the lids, infection may take place by direct transmission of the microbes or their products from

[1] Rogers. Ophthalmic Record, October, 1899.

the lids to the conjunctiva, while in such diseases as erysipelas the route may be by way of the orbital cellular tissue and optic nerve, as was observed by the writer in a well-marked case in a man seventy years of age, who died of meningitis as the result of facial erysipelas which produced orbital cellulitis. In meningitis the route may, as in the above case, be by the optic nerve, or in some instances through the sphenoidal sinus and orbital tissues.

In diseases of the respiratory tract the communication may be directly by means of the nasolacrymal canal to the conjunctival sac, or indirectly through the orbit by involvement of the frontal, ethmoidal, maxillary, or sphenoidal sinus.

In cases of serious general infection, and especially in those in which there is ulcerative endocarditis, the vessels are the usual means of transmission, and either the enormous vascular expansion of the choroid or the terminal system of the retina becomes the seat of an infectious process.

In *septicæmia,* which is often only the early stage of an infective process terminating in pyæmia, we have areas of fatty degeneration and hemorrhages into the retina. These are of grave import, as indicating the extent of the systemic invasion.

Pyæmia, having its origin in an inflammatory or necrotic process, producing infective emboli, thrombi, or hemorrhages, may result in the development of a new focus of infection in any vascular portion of the eye or orbit, the choroid and retina being most frequently the seat of this metastatic process, which may originate in an ulcerative endocarditis or an active phlebitis.

While Roth has anatomically proved that it is possible to have what might be termed a benign form of metastatic purulent retinitis which does not extend to the choroid or vitreous, it will readily be understood that a rapidly extending destructive purulent inflammation of all the structures of the eyeball is the usual result of metastatic infection of the eye in pyæmia.

In the late stages of pyæmia, even when no septic thrombus has found its way into the eye, we may have numerous retinal hemorrhages occurring immediately before death.

Malignant pustule or *splenic fever,* termed also anthrax, malignant œdema, charbon, etc., is somewhat prone to affect the skin of the lids and to cause entropion, ectropion, and infiltration of the cornea. It may even produce an orbital abscess, and severe cases in their late stages tend to cause retinal hemorrhages.

Erysipelas, classed by some writers as an affection of the skin, may properly be considered among the infectious diseases. It is not infrequently seen in the lids, though it generally originates elsewhere, and extends to the delicate skin and loose connective tissue of these structures, where it causes marked œdema, and, in rare instances, abscess and necrosis, which may lead to ectropion. Erysipelas of the lids is often accompanied by slight catarrhal conjunctivitis and some chemosis, but only in rare instances by ulceration of the cornea.

Acute dacryocystitis may be caused by erysipelas, but is not of frequent occurrence, while an exacerbation of a chronic inflammation of the lacrymal sac, or the development of an erysipelatous inflammation as a complication of a neglected dacryocystitis, is not very uncommon, and in such cases the underlying cause sometimes escapes detection, or the diagnosis of erysipelas is made when we have only an aggravated case of cellulitis due to retention in a simple purulent inflammation of the lacrymal sac.

An extension of the cellulitis of erysipelas to the tissues of the orbit is seen occasionally, sometimes taking the form of an orbital abscess and sometimes of a serous infiltration, causing evidence of pressure on the optic nerve and motor oculi. In the more severe cases there may be extension from the orbit to the brain, with resulting meningitis or sinus thrombosis, and in others exophthalmos with evidence of compression in optic neuritis, with defective vision, narrowing of the visual fields, etc., sometimes terminating in atrophy of the nerve and narrowing of the retinal vessels. Thrombosis of the central vein with retinal hemorrhage has been reported in several cases, and ptosis and paresis of the external ocular muscles, and even of the ciliary muscle, may result. Opacity of the vitreous and glaucoma have also been seen after erysipelas.[1]

A number of cases have been recorded in which an attack of erysipelas has served to hasten recovery in trachoma and diseases of the uveal tract, and this is in keeping with what has been observed occasionally in pathological processes in other portions of the body.

Whether we classify rheumatism as an infectious disease, or adhere to one of the other numerous theories as to its origin, it is certain that in some of the manifestations of the acute articular form we are dealing with an infectious process, and it is probable that in many instances the diseases of the eyes which occur during an attack of rheumatism are traceable to this cause. It is not difficult to recognize the action of an acute infection in the associated eye lesions of gonorrhœal rheumatism, nor in the embolism of the central artery of the retina which is so frequently the result of ulcerative endocarditis, but there are many cases of diseases of the eyes associated with almost imperceptible rheumatic manifestations in which the operation of an infectious process is not so apparent.

Recurring attacks of iritis, sometimes alternating with the disturbance in the joints, cyclitis, scleritis, episcleritis, tenonitis, inflammation of the bulbar portion of the oculo-orbital fascia, petechial conjunctivitis, and even paralysis of the motor oculi, may follow attacks of acute articular rheumatism.

Glaucoma, sometimes unfortunately associated with chronic iritis, is at times seen in the subjects of chronic rheumatism not necessarily articular in character, and in these cases it is difficult to avoid the

[1] Gowers, Med. Opt., p. 259.

conclusion that there is some other element in addition to the infection which we recognize in the more acute forms of the disease.

Measles in its prodromal stage is apt to be accompanied by well-marked catarrhal conjunctivitis, with the accompanying photophobia and lacrymation, and throughout the course of the disease and often for a long time after the fever has subsided, asthenopic symptoms, with weakness of accommodation, a tendency to marginal blepharitis, superficial vascular keratitis, and obstinate phlyctenular conjunctivitis and keratitis are observed. This is especially apt to be true in cases of hyperopia or astigmatism, and in such patients local and constitutional treatment generally fail to afford relief, unless the refraction error is first corrected. These manifestations are seen most frequently among those who are strumous or tubercular, though they are at times observed in patients in whom it is difficult to discover other evidences of tuberculosis. Gangrene of the lids, terminating in ectropion, has resulted in a few instances.

There have been recorded several well-authenticated cases of bilateral optic neuritis with resulting blindness, which in some instances has been permanent. They have occasionally been the result of basilar meningitis following measles, and in a few rare instances they have been secondary to purulent otitis media.

Albuminuric retinitis after measles is extremely rare.

Scarlatina is at times accompanied by conjunctivitis, but this is not so frequently a complication as in measles, and is not so valuable as an aid to early diagnosis. In the course of an attack of scarlatina, especially when it is severe, active phlyctenular conjunctivitis and keratitis may occur, sometimes resulting in extensive corneal ulcers.

Dacryocystitis is not very uncommon, and in rare instances we have orbital cellulitis with its usual consequences, even resulting in atrophy of the optic nerve, and in a few cases purulent inflammation of the lacrymal gland has been reported.

When we consider the frequency of renal complications, eye lesions due to this cause are relatively rare, though there are many recorded cases of uræmic amaurosis and amblyopia.

After describing a number of well-observed cases, J. B. Storey, quoting Foerster, makes an admirable summary which seems to be in keeping with the views of other careful observers. In all of the cases reviewed by the last-named authority "albuminuria was present, and the amaurosis occurred in the desquamation stage after a period of generally favorable symptoms. The amaurosis was ushered in by cerebral symptoms, headache, convulsions, vomiting, and stupor. It came on suddenly, was bilateral, and for a time was complete. No ophthalmoscopic lesions were detected, and the blindness gradually cleared off. There can be no doubt that these cases must be classed as uræmic."

When chronic nephritis results from scarlatina, we may, of course, at a later period have the usual retinal and nerve lesions of albuminuria. Meningitis following scarlatina may result in paralysis of

the optic nerve and, rarely, in partial or complete paralysis of one or more of the ocular muscles.

Even when no renal disease has existed and no albumin has been found in the urine, neuroretinitis has been observed.

Purulent otitis media, which occurs so frequently after scarlatina, may, by extension of the inflammation to the meninges or the development of a sinus thrombosis, lead to paralysis of the optic nerve or of the external ocular muscles, to secondary infection of the retina or choroid, or to abscess of the orbit.

Accommodative asthenopia is not uncommon after scarlatina, as after all of the exanthemata, and is much more prone to recur if, as is often the case, there is a pre-existing error of refraction.

Diphtheria may be accompanied by a most destructive form of conjunctivitis with a characteristic dense infiltration in the subconjunctival tissue; but this complication is, fortunately, of extremely rare occurrence in this country.

The ocular lesion most frequently met with, and one of extreme clinical interest, is the partial or, rarely, complete paralysis of accommodation, which comes on rapidly, and at times suddenly, from three to six weeks after the appearance of the pharyngeal lesion, and from two to three weeks after apparent recovery. It is much more frequent in children than in adults, and often appears after cases of diphtheritic infection in any part of the body, and which may be so mild as to have entirely escaped detection.

The paralysis is almost always bilateral, though there are exceptions, and it rarely affects the iris. It lasts sometimes for months, disappearing gradually, even when not treated, though the correction of refraction errors which impose an additional burden upon the ciliary muscle, and the internal administration of strychnine and iron, exert a favorable influence and shorten the period of its duration.

Complete or, more frequently, partial paralysis of the external ocular muscles, indicated by tropia and ptosis, is not very uncommon.

Neuroparalytic keratitis from involvement of the fifth nerve has been reported, and also a number of cases of concentric contraction of the visual field, with defective color vision, which latter Koenig has attributed to retinal anæsthesia.

While in rare instances hemorrhages have been observed post-mortem, near and even in the nucleus of the third nerve, it seems more probable that, as Voelckers has pointed out, the seat of the lesion is in the nerve terminations in the affected muscles. Knies has found it difficult to account for the phenomena of diphtheritic paralysis of accommodation, excepting on the theory that "a definite ptomaine is produced, and that this has a paralytic action upon accommodation, while it has no influence upon the movements of the pupil."

Variola before the introduction of vaccination was a most prolific cause of blindness, it having been estimated that in Germany 35 per cent. of all cases of blindness were due to this disease. This percentage has now been greatly reduced, ranging, according to Fuchs,

from 2 per cent. to 3.6 per cent. The same authority states that the eyes are affected in from 1 per cent. to 11 per cent. of all cases of variola, according to the statistics of various observers. Although almost every portion of the eye may be affected, vision is most frequently lost by extension of the inflammatory process from the conjunctiva to the cornea.

The skin of the lids is a frequent seat of the eruption, and it may cause œdematous swelling, hemorrhagic infiltration, abscesses, phlegmons, and furuncles or localized loss of tissue, which latter is especially serious if the pustules form on the lid margins, where they are apt to lead to trichiasis and ectropion. After the eruption has subsided there is a tendency to the formation of abscesses, boils, and disturbance of the glandular structures of the skin in other portions of the body, and this is seen also in the lids, where we have marginal blepharitis, styes, obstruction of the Meibomian glands, ectropion, trichiasis, and permanent thickening of the lids, or, if the destructive process has been extensive, great distortion or loss of lid tissue, and, in some cases, even periostitis and caries of the rim of the orbit have also been reported.

As in measles and scarlatina, the conjunctiva is apt to be congested, and we may have catarrhal conjunctivitis even when the eye is not the seat of the eruption, and there is not infrequently inflammation of the mucous membrane of the lacrymal passages.

The pustules of smallpox may form on any portion of the bulbar conjunctiva, and rarely on the palpebral mucous membrane; but their favorite location is near the corneal margin, where they are smaller than when seen on the skin, and present the appearance of conjunctival phlyctenules. There is severe inflammation, with chemosis and active secretion, as in purulent conjunctivitis, and secondary corneal infection is very apt to follow quickly, with hypopyon and all that such a destructive process means.

Some authorities deny that the primary eruption is ever seen upon the cornea, and occurring, as it usually does, many days after the appearance of the cutaneous eruption, or even during convalescence, it is probable that the corneal infection is a secondary process which, as Knies has suggested, may be held in check by the diligent use of aseptic and antiseptic treatment.

Even when there is no conjunctivitis nor evidence of eruption on the eyeball, we may have corneal involvement, due apparently to local infection by morbid material circulating in the blood. Indeed, this is by some authorities said to be the most frequent type of corneal involvement (Story). It occurs usually as a late manifestation, and is apt to be associated with or followed by such serious complications in other organs that a fatal termination is not uncommon.

Iritis, usually as a part of an anterior uveitis involving a low grade of cyclitis, vitreous opacities, and more or less involvement of the choroid, is not an unusual manifestation during the late stages of variola, and isolated areas of choroiditis are sometimes, although rarely, observed.

A few cases of neuroretinitis and hemorrhages into the optic nerve have been described, and more would probably be reported if ophthalmoscopic examinations were more common.

Secondary nephritis is comparatively rare, but is sometimes present, and may be accompanied by uræmic amaurosis and albuminuric retinitis.

Vaccination, like other infectious processes, may cause disease of the eyes. Eczema of the face and phlyctenular disease of the conjunctiva and cornea, which sometimes follow or accompany vaccination, are of comparatively slight importance in healthy patients, but in children and others of feeble constitution they may pursue a tedious course. Accidental vaccination of the lids, conjunctiva, and cornea has been reported many times, and, whether from a vaccine pustule or from a lymph tube, produces a most serious infection. If the cornea is affected, it is apt to become the seat of active infiltration, strongly resembling a vaccine pustule.

While the transmission of other diseases by vaccination is not so common as is generally supposed, it is by no means impossible for such a result to follow when the conditions are favorable, and when syphilis is transmitted we may have iritis and the other usual manifestations of that disease in the eye.

Varicella rarely causes eye complications, but one instance is recorded of iritis accompanied by hypopyon.

Typhoid Fever. A degree of hyperæmia or conjunctivitis may occur in typhoid as in other fevers, and phlyctenular conjunctivitis and keratitis are not uncommon during convalescence. Keratomalacia may result in serious cases, and, with the profound and prolonged somnolence which is sometimes a characteristic of the late stages, we often have xerosis of the cornea accompanied by infection from exposure. A gangrenous inflammation of the lid which rarely makes its appearance is apt, if the patient survives, to lead to ectropion.

While not very common, various affections of the uveal tract are sometimes present, as anterior uveitis, plastic iritis, cyclitis, choroiditis, and choroidoretinitis, with their accompanying changes in the vitreous and later in the lens.

The general muscular weakness which often characterizes the period of convalescence after typhoid fever manifests itself in the eyes as prolonged and sometimes extreme weakness of accommodation, producing much more marked symptoms if astigmatism or hypermetropia is present. In addition to accommodative we often have muscular asthenopia, especially if a manifest or latent imbalance of the external ocular muscles is present. True paralysis of the external muscles, with deviation or ptosis, is rarely, though sometimes seen as a consequence of typhoid fever, and, as Knies has well said, should suggest a careful study of the urine.

The various diseases of the brain and spinal cord which are occasionally seen as sequelæ of typhoid fever may be accompanied by

characteristic lesions of the optic nerve and retina. Either during or after the attack we may have retrobulbar neuritis, followed in some instances by atrophy; and neuroretinitis with macular hemorrhage has been reported, as have also cases of amaurosis and hemianopsia, apparently due to cortical disease.

Typhus fever is known sometimes to cause catarrhal conjunctivitis, inflammation of the uveal tract, and other lesions similar to those observed in typhoid fever, but few instances of such complications have been published.

Typhus recurrens or *relapsing fever*, like typhoid and other fevers, causes more or less catarrhal conjunctivitis, and may be accompanied by phlyctenular conjunctivitis and keratitis. It leads also to degeneration and weakness of the muscular system, which result in accommodative and muscular asthenopia, especially noticeable during convalescence. Directly or indirectly, the poison of this fever may so affect the visual centres, the tract, or the optic nerve, as to cause temporary or permanent blindness; but this is of rare occurrence when compared with the lesions of the uveal tract which often make their appearance as late manifestations or, more properly, as sequelæ of typhus recurrens. A diffuse inflammation of the uvea, and especially of the ciliary body, varying in intensity from little more than a mild hyperæmia to an active purulent cyclitis, may be found, and this complication may result in only a slight clouding of the anterior portion of the vitreous, which will clear away completely in the course of a few weeks, or it may lead to an active choroiditis and retinitis, with purulent infiltration of the vitreous and subsequent phthisis bulbi. It frequently assumes the form of an anterior uveitis (serous iritis), and sometimes is accompanied by hypopyon. One or both eyes may be affected, and while its occurrence as a complication is not necessarily dependent upon the severity of the general disease, ocular lesions are of far more frequent occurrence in some epidemics than in others.

Knies, who has written quite fully on this subject, points out the very important fact that analogous changes probably take place in the pia mater, but those appearing in the uveal tract, owing to its intimate relation with the retina and vitreous, are much more apparent.

While occlusion of the pupil by the exudate from the ciliary body and iris and dense opacities of the vitreous, or even secondary detachment of the retina, may result in severe cases and lead to blindness, the prognosis is not generally unfavorable, as the ring of faint vitreous opacities is, in a large proportion of cases, sufficiently absorbed to admit of good vision.

Malaria may modify various diseases of the eyes which are due to other causes and give a periodicity to their manifestations which yields to the action of quinine. It may produce diseases of other organs which indirectly affect the eyes, or it may be the direct cause of disease of the eyes.

Periodic trigeminal neuralgia, generally of the supra-orbital branch, is quite common, and occasionally true ciliary neuralgia is due to this cause.

Conjunctivitis and plastic iritis have been described, but are rare, while keratitis is of comparatively frequent occurrence. Neuro-paralytic and interstitial keratitis are seen at times, but a more common form is what is termed dendritic keratitis, which presents itself as a superficial infiltration with narrow irregular streaks radiating from it, sometimes assuming the form of a serpiginous ulcer. When this form of corneal infiltration is found associated with tenderness on pressure over the supra-orbital nerves, quinine in full doses is indicated, in addition to such other local and general treatment as may be found appropriate. Herpetic corneal eruptions are also sometimes seen after prolonged attacks of malarial fever.

Muscular paralysis has rarely been noted, and temporary and even permanent amblyopia and amaurosis are of not very infrequent occurrence. The amblyopia may be accompanied by scotomata or contraction of the periphery of the visual field. Permanent defects of the visual field are due to partial atrophy of the optic nerve, and occur only in the more severe cases of malarial poisoning.

As Knies has pointed out, a large proportion of cases of malarial amblyopia and amaurosis are probably of central origin, and when occurring at the onset of the attack he considers them to be the result of ptomaine-poisoning. A few instances of malarial hemianopsia have been reported. Torpor of the retina amounting to night blindness occurs not infrequently in chronic malarial poisoning; and Baas has observed a case of blue vision appearing with a regular periodicity, with other evidences of intermittent fever, and disappearing after the administration of quinine.

The prognosis of malarial amblyopia and amaurosis is generally not very grave, although in rare instances serious defects of vision may remain. By far the most important lesions resulting from malarial infection appear in the uveal tract, retina, and optic nerve. Grave cases of malarial poisoning are sometimes accompanied by intra-ocular hemorrhages which may appear at the onset of the disease, but are more common in the later stages; and Poncet has established the fact that the accompanying changes in the choroid and retina are due to obstruction of the capillaries and smaller vessels by pigment flakes and other abnormal elements in the blood. He found on autopsy "œdema, hemorrhages, lesions of the walls of the vessels, and thrombosis and small inflammatory foci with and without pigment," etc.

The vitreous opacities, exudative retinitis and choroiditis, and hemorrhage or exudation into the optic nerve may result in serious defects of vision, but fortunately such results are of infrequent occurrence.

Influenza, which may be an important factor in the causation of diseases of almost every organ in the body, makes no exception of

the eye; but while in its erratic course it may produce an almost infinite variety of eye symptoms, there is little uniformity in their character, and few of them appear with such frequency as to make them typical manifestations of this disease.

Conjunctival hyperæmia, with more or less lacrymation and photophobia, is of extremely frequent occurrence as an early manifestation, and, especially when the nasal mucous membrane is actively involved, we may have an acute catarrhal or mucopurulent conjunctivitis. Pre-existing conjunctivitis in its various forms is apt to be aggravated, œdema of the conjunctiva may be present, and sometimes we have a number of minute extravasations of blood into the conjunctiva as a result of the attacks of severe coughing.

Œdema of the upper lids is sometimes present in the period of convalescence, and rarely inflammation of the lacrymal gland has been known to develop.

A deep abscess of the lids is seen at times as a late manifestation, and has been attributed to involvement of the frontal sinus; and hordeola are of not infrequent occurrence, while embolic orbital suppuration has been recognized in rare cases.

Dacryocystitis is apt to develop if a stenosis has already existed.

Paresis of accommodation as a sequela of influenza is not very uncommon, but true paralysis is rare, and nuclear or peripheral paralysis of the extrinsic ocular muscles, although sometimes seen, is of very infrequent occurrence, resulting in tropia or ptosis. Blepharospasm is found more frequently. The neuralgic pain and tenderness of the eye and its surroundings are probably due in large part, as Knies has suggested, to a low form of orbital periostitis, resulting from disease of the mucous lining of the frontal sinus and other periorbital cavities.

Phlyctenular keratitis, at times associated with phlyctenular conjunctivitis, has occasionally been reported; and also punctate superficial keratitis and herpes of the lid and cornea, in the latter situation sometimes assuming the form to which the term dendritic has been applied.

Various affections of the uveal tract have been reported, although it is not so frequently involved as we would expect when we remember how often influenza occurs in those of such age as to have vulnerable bloodvessels.

Judging by his personal experience, the author would say that a low grade of hyalitis is the most frequent manifestation of involvement of the uveal tract as a sequela of influenza, although hyperæmia of the iris, plastic iritis, embolic iridocyclitis, purulent uveitis with hypopyon, and even panophthalmitis, have been reported, and cases have been described which resulted in the development of a purulent tenonitis.

Non-septic embolism of the central retinal artery has been reported in a few cases, but in so rare a complication we should bear in mind the possibility of a coincident cause. Although not of frequent occur-

rence, a number of instances of optic neuritis have been recorded, some resulting in partial, and others in complete atrophy of the optic nerve; and Berger mentions several cases of temporary amaurosis which were probably due to the toxic effect of the ptomaines developed during an attack of influenza.

Glaucoma has been known to be precipitated by an attack of influenza, but it is probable that defective bloodvessels and other unfavorable conditions contributed to this result.

Whooping-cough. Conjunctival injections, photophobia, and lacrymation are not uncommon in the early stages of pertussis, and phlyctenular disease of the conjunctiva and cornea, sometimes occurs as a sequela.

But while we occasionally meet with evidences of disease in the eyes which may be said to result from indirect action of the infective agent, by far the larger proportion of the ocular manifestations are of purely mechanical origin, and result from the increased intravascular tension due to the spasmodic cough. Hemorrhages in the conjunctiva and lids are absorbed within a few weeks, leaving no trace, and small hemorrhages in the orbit may escape notice, while large ones, which fortunately are rare, may cause exophthalmos.

Paralysis of the ocular muscles may occur, with consequent strabismus or ptosis, and is due to hemorrhages.

Convergent squint, the earliest appearance of which so frequently follows the various infectious diseases of childhood, seems to occur with great frequency after whooping-cough, though it is seldom due to paralysis, and is associated usually with, and in a large measure dependent upon, hypermetropia.

Mumps. In addition to œdema of the lids, conjunctivitis, keratitis, and rarely iritis, mumps may, like other infectious diseases, occasionally be the cause of serious congestion or even inflammation of the optic nerve and retina, or primary optic nerve atrophy. Paresis of the ocular muscles and of accommodation may result, and metastatic iridocyclitis has been reported.

Like the testicle, the lacrymal gland may be the seat of an inflammation apparently due to the same sources of infection as that which produces the parotitis, or at least secondary to inflammation of the parotid gland.

Cholera. With the sudden onset of emaciation and great loss of blood serum which are characteristic of it, cholera produces a striking change in the expression of the eyes, and often is accompanied by profound lesions in the deeper structures which, to a limited degree, may be seen in severe cases of other forms of disease of the gastro-intestinal tract, such as cholera infantum and ordinary cholera morbus. The cyanosis and shrinking of the eyeballs into the orbit, with shrinkage of the lids due to absorption of fluid, and the weakness of the orbicularis palpebrarum muscle, with its resulting imperfect closure of the palpebral fissure, give to these cases a most striking

and alarming expression. There is a lack of secretion, resulting in great dryness of the conjunctiva and cornea, and it is said that neither the great pain nor the contact of irritating substances is sufficient to provoke lacrymation.

While by a great effort the patient can close the lids, they habitually remain partially open, exposing the inferior bulbar conjunctiva and lower portion of the upturned cornea, which often becomes the seat of an ulcer and, if the patient recovers, of a leucoma. The exposed conjunctiva may be merely injected or in more severe cases inflamed and xerotic. A peculiar form of irregular grayish patches, sometimes isolated and sometimes confluent, is seen about the corneal border in severe cases. They are attributed by Knies to choroidal hemorrhages shining through the thinned sclera, and are of grave prognostic significance, as are also the spontaneous hemorrhages which at times appear beneath the conjunctiva.

Myosis seems more common than mydriasis, though the latter is sometimes observed. Active reaction to light is a favorable prognostic sign, while even in apparently mild cases immovable pupils almost certainly indicate a fatal termination. Owing to the weakness of the cardiac muscle and the marked lowering of the general intravascular pressure when the disease is in the algid stage, great variations in the appearance of the fundus are to be noted on light digital pressure during an ophthalmoscopic examination. An interruption of the blood current and intermittent circulation is sometimes observed resembling that which accompanies restoration of the normal movement after an embolism of the central retinal artery has been displaced.

Yellow Fever. While many cases of yellow fever exhibit no characteristic eye lesions, we may have intra-ocular hemorrhage with its serious consequences in impaired vision, or uræmic amaurosis associated with cerebral symptoms, and in either case the prognosis is grave.

Syphilis in all of its stages may affect the eyes, and while the course it pursues in this organ depends much upon the virulence and the stage of the general infective process at the time the eye is involved, and upon the part affected, no portion entirely escapes its ravages, although the uveal tract, because of its great vascularity, seems to be its more usual point of selection in the acquired form, and iritis, cyclitis, choroiditis, and hyalitis are its more common ocular manifestations.

Syphilis does not very frequently produce absolute loss of sight, Mangus having found 2.2 per cent. of blindness due to this cause; but, especially in large cities, where syphilis is more or less common, it is a frequent cause of marked impairment of vision. Alexander, from a study of the statistics of eight German ophthalmological clinics, has estimated that 2.16 per cent. of diseases of the eye are the result of syphilis. This would probably be a high estimate if it applied to both hospital and private practice, at least in America.

Our attention will first be directed to acquired syphilis in its various forms, the subject of congenital or hereditary syphilis being considered later.

The initial lesion may be found upon some portion of the eye or its appendages, and, if we except the genital organs and the mouth, this is relatively a frequent point of primary infection. Hard chancres have been found upon the lids, especially at the free margin where the cutaneous and mucous surfaces join, upon the palpebral conjunctiva, the plica semilunaris, the caruncle, in the retrotarsal folds, very rarely upon the bulbar conjunctiva, and even upon the cornea. The most common locations seem to be the caruncle and free margin at the inner canthus and along the lower lid. Infection may be the result of a kiss from an individual with a mucous patch on the mouth, or from contact of infected fingers. Instances have been recorded in which attendants and physicians were infected in the latter manner while treating syphilitic patients. The characteristic hard sore develops, first presenting the appearance of a pimple, which later breaks down into a shallow ulcer with rounded edges and an indurated base. The enlargement of the lymphatic glands at the angle of the jaw and in front of the ear, which is quite characteristic and sometimes extensive, should aid in distinguishing a chancre from simple ulcerations due to other causes. Though recovery may be quite complete, the induration often continues for many months, which is apt to be misleading, unless the case is carefully observed. Hard chancre of the lids may be confused with epithelial cancer, and chancroid may be found in the same locations, making it necessary at times to await the development of secondary manifestations to determine the cause.

Secondary manifestations in the form of roseola or acne-like eruptions may affect the skin of the lids and cause loss of the lashes, and characteristic ulcerations of the tertiary stage may appear later.

Rarely there are mucous patches on the palpebral and even upon the bulbar conjunctiva, and gummy tumors of the ocular conjunctiva have been seen. de Schweinitz mentions an inflammation of the conjunctiva assuming the form of a catarrhal conjunctivitis or follicular trachoma, developing "in an anæmic and rather colloid-looking conjunctiva," which yielded only to antisyphilitic treatment.

An interstitial or gummatous inflammation of the lacrymal gland has been seen in rare instances, and syphilitic disease of the periosteum and secondarily of the bony walls and orbital contents is of relatively frequent occurrence, causing protrusion or fixation of the globe and all of the evidences of exudative disease or tumor in this region, sometimes going on to suppuration and the formation of fistulæ.

A careful study of the history and other manifestations of syphilis is necessary to determine the true nature of such cases, and a positive

diagnosis is sometimes made only after the symptoms have yielded to alterative treatment.

Syphilitic disease of the nose not infrequently leads to disease of the mucous lining, periosteum, and bony walls of the lacrymal passages, producing dacryocystitis, and later firm stenosis or bony occlusion.

The uveal tract, as has been stated, is the point of selection for syphilis of the eye, especially in the secondary stage, and, owing to the intimate relation of the choroid and retina, and the dependence of the vitreous humor upon the uveal tract for its nutrition, we are apt to find an extensive inflammation of one portion, involving the others to a greater or less degree. This is more especially true of chronic diseases, in which prolonged impairment of nutrition plays an important part; but it is probable that the difference is only one of degree, and even in an acute plastic iritis we have, to some extent, a secondary involvement of the whole uveal tract and incipient changes in the retina and vitreous.

Friedenwald[1] has demonstrated that in every case of iritis a properly conducted examination will reveal deposits upon Descemet's membrane, which is the only part of the uveal tract, in addition to the iris, in which such an investigation can be satisfactorily made during the active stage of iritis.

Plastic iritis as an early manifestation of general syphilis commonly makes its appearance between the second and ninth month after the initial lesion, though it may appear many months later. Both eyes may be attacked simultaneously, though it often happens that prompt and vigorous treatment will prevent involvement of the second eye. Authorities differ as to the frequency of iritis in syphilis, some placing it as low as 0.42, while others state that 5.37 per cent. of syphilitic patients have iritis. Alexander, who has made an extensive research into the statistics of syphilis, places the proportion of cases of iritis in which syphilis can be assigned as the cause at from 30 to 60 per cent. Simple, plastic iritis of syphilitic origin has no characteristics which of themselves prove its etiology, and the diagnosis of syphilis must be based upon other evidence; but in a certain proportion of cases, variously estimated at 15 to 20 per cent., we have "iritis gummosa, papulosa, or condylomatosa, which is sufficiently typical to afford a fair basis for a diagnosis." Often associated with a small hypopyon, we find in these cases a yellow or dull orange-colored nodule, generally situated near the lower margin of the pupil and surrounded by a narrow zone of red. In the later stages of severe syphilitic infection, we sometimes have large gummy tumors, almost filling the anterior chamber. With the exception of the crystalline lens, any portion of the eye may be the seat of a syphilitic lesion taking the form of a gumma or interstitial inflammation accompanied by atheromatous thickening of the intima of

[1] Arch. d'Ophtalmol., vol. xxv. p. 191.

the bloodvessels, which often results in occlusion; and as these vascular lesions do not entirely disappear under treatment, they sometimes lead in the eye, as they do in the brain, to the impaired nutrition, hemorrhages, etc., which account for many of the indirect late manifestations of syphilis.

It is of great importance to bear in mind that antisyphilitic treatment can have little effect upon such late lesions, and it by no means follows that a lesion of long standing is not of syphilitic origin because it fails to respond to antisyphilitic treatment. Anterior uveitis of the form sometimes spoken of as serous iritis or descemetitis may be found as a result of syphilitic infection of an asthenic type, but such cases are of rare occurrence.

Syphilitic cyclitis and choroiditis of almost every degree of severity appear with relative frequency, and the latter is associated usually with retinitis and hyalitis. While a large proportion of cases of choroiditis have their origin in syphilis, it is the opinion of the writer that the text-books on ophthalmology have given undue weight to this disease as an etiological factor; and though stress has been laid upon certain characteristics as indicating such a cause, there are no infallible ophthalmoscopic appearances which, taken alone, will warrant a diagnosis of syphilis. In deference to the opinion of some high authorities, it should, however, be stated that disseminated choroiditis, and especially symmetrical choroiditis areolata, and the existence of large quantities of fine, dust-like vitreous opacities, are of not infrequent occurrence, and are by many regarded as pathognomonic of syphilis.

Inflammation of the choroid, which may be disseminated or areolar in type, is sometimes mild, but more frequently severe in character. When it appears in the earlier stages of the infection and is treated promptly and thoroughly, syphilitic choroiditis often yields readily; but, as in other structures, in the later stages of the disease, when extensive changes have taken place in the vessel walls, we cannot expect prompt response to alterative treatment, however active it may be.

In association with syphilitic cyclitis and iritis, syphilitic choroiditis not infrequently results in entire loss of sight, detachment of the retina, softening, and phthisis bulbi. It is almost invariably accompanied by more or less extensive retinitis, and is properly designated as choroidoretinitis. Unless the macular region is involved or vitreous opacities are present, choroiditis and choroidoretinitis, even though quite extensive, are apt to escape the attention of the patient; but when central, the earlier stages are manifested by a variety of more or less pronounced subjective symptoms, evidently due to irritation and disturbance of the outer layers of the retina by the diseased choroid. Photophobia, sparks before the eyes, micropsia, metamorphopsia, subjective colored vision, etc., soon give place to diminished visual acuity, torpor of the outer layers of the retina, and often a positive scotoma, followed by general clouding due to vitreous dust

or larger opacities. As the disease progresses and produces more and more disturbance of the pigment epithelium, we have a variety of ophthalmoscopic pictures of retinitis, some to a degree resembling retinitis pigmentosa, in which at the point of adhesion between the choroid and retina the pigment of the former emigrates into the latter, some due to areas of dense retinal opacity, and others in which the characteristic specific exudation has produced changes in the appearance of the retinal vessels. The dust-like opacities of the vitreous, sometimes spoken of as pathognomonic of syphilis, the writer has found in many cases which were undoubtedly due to other causes. While sometimes appearing as early as six months after the primary infection, syphilitic choroidoretinitis is usually a late manifestation.

The prognosis as to vision must depend largely upon the stage at which treatment is undertaken and the part of the retina involved. If the macular region is the seat of the disease, we almost invariably have serious impairment of vision.

The cornea, sclera, and oculo-orbital fascia may be the seat of acquired syphilis, but involvement of these structures is of rare occurrence, and is generally secondary to disease of other parts of the eye.

Syphilitic optic neuritis, not secondary to disease at the base of the brain or in the other structures of the eye, though of rare occurrence, has been observed, and simple double atrophy of the optic nerves is said to occur at times unaccompanied by spinal symptoms.

An almost infinite variety of lesions in the cortex, at the base, and, indeed, in every portion of the brain, may result from syphilis, manifesting themselves in the production of cortical symptoms, such as hemianopsia, etc., and by their effect upon the optic nerve and retina and the motor and sensory nerves of the eyes. This subject is more properly dealt with in the section on diseases of the nervous system, but a brief review will here be given of the more important considerations in connection with syphilitic paralysis of the ocular muscles.

The paralysis is usually peripheral. The muscle itself may be the seat of the disease, or a gummatous growth may develop in the neighborhood of the nerve as it passes through the orbit or at the base of the brain, or a specific lesion may affect the nuclei or the point of origin of the nerve in the third or fourth ventricle or in the aqueduct of Sylvius.

According to Alexander, 59.4 per cent. of paralyses of the ocular muscles are due to syphilis. They are usually late manifestations, rarely appearing during the first six months, and while they may develop rapidly or come on very gradually, they usually respond to treatment rather slowly in those cases which prove to be curable. Naunyn, quoted by Knies, reports 70 per cent. of recoveries, but states that if improvement does not manifest itself within two weeks under vigorous treatment there is no hope of recovery. Other authorities, however, encourage perseverance for a much longer period. Relapses

are uncommon if the treatment is maintained for a sufficient length of time.

While in some instances it may be the first symptom of syphilis, and therefore of great diagnostic importance, paralysis of an ocular muscle is found more frequently in association with other evidences of this disease. According to Knies, the isolated paralyses "are due either to neuritis and perineuritis of the nerve roots and at the base of the brain, or they are nuclear in origin; other causes are exceptional."

Unilateral paralysis of the branches of the third nerve supplying the sphincter of the iris and the ciliary muscle is not uncommon. Paralysis of the fourth and facial nerves is rare, representing about 1 to 2 per cent. each of all cases due to syphilis, while the sixth is affected in about 25 per cent., and the oculomotor in 75 per cent. Paralysis of the fourth and seventh nerves when present is apt to be associated with paralysis of the third or sixth.

In considering the question of the presence of syphilis in a given case of disease of the eye, it should be remembered that we are largely dependent upon collateral evidence, as the cases are very rare in which the ocular lesions taken alone are pathognomonic. Therapeutic measures as a means of diagnosis are at times of great value; but while, on the one hand, many non-syphilitic lesions are favorably influenced by the use of the so-called alterative treatment, it is a well-recognized fact, which has already been referred to, that there are certain late manifestations or results of syphilis, especially in the nervous system and eye, upon which, owing to secondary changes in the vessel walls or the non-vascular nature of the tissues affected, the iodides and salts of mercury appear to exercise no influence. Patients whose tissues have undergone such changes, though no longer, properly speaking, the subjects of active syphilis, may develop erratic attacks of various forms of ocular paralysis somewhat resembling true syphilitic paralysis, but more like those seen in multiple sclerosis. These symptoms will not yield to antisyphilitic treatment.

Congenital Syphilis. Owing possibly to attenuation of the specific poison in transmission through the tissues of the mother, or to a process of selection by which only the less serious cases survive, congenital syphilis is usually a milder disease in its effects upon the eyes than is the acquired form, and it runs quite a different course, although it is often far more obstinate and unyielding to treatment. Intra-uterine syphilis, on the other hand, is said to run its course much more rapidly, the fœtus in many instances dying of tertiary syphilis.

As in the acquired form, it is the uveal tract that is the point of selection in congenital syphilis, and while diffuse interstitial or "parenchymatous" keratitis is its most common and easily observed manifestation, this is what has been termed an emigration keratitis, and is secondary to and in association with other lesions of the uveal tract.

Choroiditis in its various forms, and mild or most severe plastic iritis, iridocyclitis, and iridochoroiditis develop in some instances, and are accompanied by softening of the eyeball.

As the various structures of the eye may be primarily or secondarily affected, either *in utero* or in early childhood, it will be readily understood that opacities of the cornea, occlusion of the pupil, cataract, opacities of the vitreous, and atrophy of the choroid, retina and optic nerve are not infrequently met with in congenital syphilis. Congenital atrophy of the optic nerve or neuritis is not infrequently the result of intra-uterine meningitis of syphilitic origin.

As in acquired syphilis, we sometimes find in the congenital form that periostitis or caries of the walls of the orbit leads to most troublesome symptoms, among which persistent occlusion of the nasal duct is of not infrequent occurrence.

Paralyses of the ocular muscles may also occur, but are rare. By far the most frequent ocular manifestation of congenital syphilis, as has been stated above, is diffuse interstitial keratitis, and while this may be due to other causes, its presence should always lead to a careful investigation. The evidences of the inheritance of a syphilitic taint are too well known to require consideration here; but while certain Continental writers have been inclined to accept with many qualifications the indications pointed out by Hutchinson in the peculiar formation of the incisor teeth, and while other diseases doubtless may, in rare instances, produce similar changes, the writer has found them so frequently associated with congenital syphilis that when present he regards them as of the greatest diagnostic value.

As in all late manifestations of syphilis, many of the symptoms of the hereditary form of the disease, especially those appearing after infancy, are due to secondary changes not the immediate result of the infection, and it is a mistake to expect results from alterative treatment, such as may be obtained in the earlier years of acquired syphilis. Too great persistence in the use of the iodides and mercurials in such cases may do great harm; but, on the other hand, the writer is convinced by his own experience that judgment and discrimination should be exercised in this as in all questions of therapeutics, and the above principle must not be too slavishly adhered to, as it not infrequently happens that brilliant results are obtained by the discreet use of these remedies even in subjects of congenital syphilis who are no longer young.

Leprosy often has a very long period of incubation, and the primary lesion is apt to escape attention. It is very prone to affect the lids and brows; the former, according to Lopez, being involved at some time in the course of almost every case of this disease. Eklund has stated that infection often occurs in the conjunctival sac from the use of towels, and the bacilli of leprosy, resembling those of tuberculosis, have been found in the tears.

The eyebrows and lids may be the seat of anæsthetic patches or nodules, which lead to loss of the hair of the brows and the eyelashes,

and when ulceration takes place we are apt to have ectropion or entropion. Lopez calls attention to the occurrence of lagophthalmos due to involvement of the terminal motor nerve elements distributed to the orbicularis muscle.

In the conjunctiva leprosy produces anæsthesia, followed by chronic conjunctivitis; and pterygia and tubercles may develop, which frequently lead to keratitis and pannus, especially of the lower half of the cornea. The tubercles of the conjunctiva may terminate abruptly at the corneal margin and lead to secondary clouding and other degenerative changes, or the deeper layers of the cornea may be the seat of the leprous tubercles. In a later period of the disease we may have distinct involvement of the cornea, resembling interstitial keratitis, and ulcers are not uncommon.

Involvement of the iris may be secondary to keratitis, but is sometimes an early manifestation, and may take the form of an acute iritis or the development of grayish nodules or tubercles, especially near the periphery in the lower half. If the disease reaches the iris, we are apt to find vitreous opacities, cyclitis, and choroiditis, with secondary cataract; and in the late stages there may also be involvement of the choroid and retina. The progress of leprosy is slow, and the fact that small nodules in the iris have been known to disappear under treatment is referred to by Knies.

Tuberculosis. While primary tubercular infection of the eye is sometimes observed, it is of not very frequent occurrence, but secondary involvement is far more common.

Lupus, which is generally conceded to be of tubercular origin, may appear upon the lid as an ulcerated area, with red granular patches, and later may extend to the conjunctiva, and finally to the eyeball, causing its destruction. The conjunctiva, if the surface is broken, may be the seat of primary infection. This, although unusual, has been well established in a small number of cases. In some instances we have tubercular infection of the conjunctiva, which in appearance for a time resembles trachoma, although its later course, the fact that it will not yield to ordinary treatment, and its frequent association with nasal and laryngeal tuberculosis will determine its character. More frequently we find "caseating" ulcers with irregular raised edges, sometimes covered with grayish nodules, showing a tendency to slough. This may be associated with considerable swelling of the lids; there is enlargement of the lymphatic glands of the corresponding side, and the patients are apt to show other evidences of tuberculosis.

The diagnosis may be made more certain by the microscopic examination of small pieces of tissue or particles of the cheesy contents of the nodules or by inoculation.

Tubercles of the iris containing the characteristic bacilli, and also giant cells, are found occasionally as an apparently primary manifestation of the disease. According to Eyre, it does not usually implicate the cornea until late in the course of the disease, and the iris still later.

Involvement of the iris, as well as other tubercular diseases of the eyes, is more common in children than in adults.

In cases of general tuberculosis the uveal tract and the choroid especially is sometimes involved, the ophthalmoscope revealing yellowish-white spots, often appearing to be not more than one or two millimetres in diameter, and difficult to detect. Large tubercular tumors resembling sarcomata are seen also at times. Choroidal tubercles visible with the ophthalmoscope, if seen at all, so often appear in the late stages of the disease that they are of only slight diagnostic value.

The development of tubercles within the eye is often associated with reduced intra-ocular tension, but in a few instances increased tension has been noted, and Lubowski has reported one case of absolute glaucoma.

DISEASES OF THE SKIN.

The conjunctiva and superficial layers of the cornea being continuous with and anatomically and embryologically closely related to the skin, it is only natural to infer that many of the diseases of the latter should present themselves in a modified form in these portions of the eye and often lead to complications in the deeper structures. This inference is found to be correct, especially in the case of such diseases as eczema and herpes; while diseases such as lupus and epithelioma frequently extend from the lids into the eye; and the parasitic and other cutaneous affections assume a slightly modified form in the lids, owing to the fact that the skin here differs in some respects from other portions of the integument.

Eczema, which assumes such a variety of forms in various portions of the cutaneous surface, frequently affects the eyes; and here, as elsewhere, it presents itself in forms that differ so widely as to be somewhat confusing to one of limited clinical experience. It may affect the lids, conjunctiva, or cornea, and while of very frequent occurrence in childhood, is more rare in patients of advanced years, although in those subject to gout and rheumatism it is sometimes associated with conjunctivitis, and proves most obstinate and distressing. In adults it is apt to present itself on the surface of the lid in the form of eczema squamosum, but it is among children that we meet with large numbers of cases of this disease, and here, while the surface of the lids often is affected, it is the conjunctiva and cornea which deserve most careful study.

Owing probably to the ease with which the conjunctival and corneal epithelium is broken, we seldom meet with true vesicles, but points of infiltration assuming the form of pinkish-yellow elevations in the bulbar conjunctiva, and grayish or grayish-yellow infiltrations in the cornea, are very common. These are generally spoken of as phlyctenulæ, and often are found in association with eczematous

disease of the mucous membrane of the nose, and especially in tuberculous or "scrofulous" children with eczematous eruptions about the nose and mouth, and adenoids, hypertrophied tonsils, and enlarged postcervical glands.

Herpes zoster of the integument of the lids may occur under conditions favoring its development elsewhere on the face, especially when vesicles are found on the side of the nose, and herpes of the cornea is not very uncommon. The latter assumes the form of herpes zoster ophthalmicus, sometimes corresponding to what is termed neuroparalytic keratitis, and by secondary infection may lead to extensive and dangerous ulceration. The neuralgic and burning pain induced by herpes zoster may precede and continue for a long time after the eruption has disappeared.

Herpes vulgaris or **febrilis,** which is a more common disease of the cornea than herpes zoster, is often found in association with catarrhal diseases of the digestive or respiratory tract, and is characterized by the formation of one or more small vesicles which often are broken before their character is recognized. The surface of these ulcers may be anæsthetic, but not the surrounding portions of the cornea, and while they often pursue a sluggish course, infection from the conjunctival sac frequently occurs, and may lead to the development of a destructive serpiginous ulcer. What is termed *keratitis dendritica* or *keratitis ramiformis,* is by some authorities classified as a variety of herpes corneæ, although it is more probable that it is dependent for its characteristic form upon a special micro-organism.

Seborrhœa, which is characterized by the development of acne in other portions of the face, when it involves the sebaceous glands of the borders of the lids produces hordeolum or stye. The large size of the glands in this region, the nature of the surrounding tissues, and the ease with which infection may take place, account for the difference between hordeolum and acne as it appears in other portions of the integument.

Favus, lichen ruber, acne rosacea, milium, and **erythema multiforme,** all may appear on the skin of the lids, but they present few characteristics differing from those seen when they are found in other portions of the face.

Furuncle is occasionally seen in the upper lid, causing marked tumefaction and redness.

Molluscum contagiosum may appear on the lids, and Mittendorf has observed two epidemics occurring in a hospital.

Elephantiasis arabum is sometimes confined to the lids, though generally appearing with a similar condition in other parts of the body.

Pemphigus of the conjunctiva is occasionally observed, and **ichthyosis,** in addition to causing shortening of the lids, may extend to the conjunctiva and eyeball.

Phthiriasis of the edges of the lids may sometimes be recognized by the presence of nits upon the eyelashes, and sycosis and other parasitic affections are occasionally found in this region.

Purpura may provoke small hemorrhages in the skin of the lid and under the conjunctiva and into the retina, as well as in other portions of the body.

Impetigo and **psoriasis** sometimes invade the skin of the lids, the conjunctiva and conjunctival portions of the cornea.

Urticaria is found occasionally on the lids, and iritis and paralysis of accommodation may be present as a result of the toxic element causing the skin eruption.

Alopecia of the brows and lashes, often complete, may be found either with or without manifestations elsewhere.

Lupus as a local manifestation of tuberculosis is more properly treated under the head of infectious diseases, where erysipelas is also considered.

Pellagra, due to ingestion of a fungus of maize by poorly nourished individuals, according to Rampoldi, produces torpor of the retina, retinitis pigmentosa, atrophy of the optic nerve, disappearance of the choroidal pigment, marantic ulcers and necrosis of the cornea, and opacities of the lens and vitreous.

DISEASES OF THE BRAIN AND SPINAL CORD.

Cerebral Hyperæmia and Anæmia. With the exception of certain conditions accompanied by prolonged venous hyperæmia or congestion of the brain, as is sometimes seen in epilepsy of long standing, we look in vain to the ophthalmoscope for information as to the condition of the cerebral circulation, although it is of the utmost value in studying diseases of the bloodvessels, as in such conditions as arterio-capillary sclerosis, etc., pronounced hyperæmia or anæmia of the brain may coexist with a normal fundus, and the existence of hyperæmia of the retina may not be accepted as proof that a corresponding condition will be found in the brain. Those unfamiliar with the physiological variations in the appearance of the normal fundus not infrequently fall into serious error in the inferences they draw as to the condition of the cerebral circulation. Indeed, the author has known high authorities on diseases of the nervous system, but with limited experience in the use of the ophthalmoscope, to be entirely misled, and to base a most grave prognosis upon the apparent congestion of the retinal vessels familiar to every experienced ophthalmologist as one of the characteristics of high degrees of hypermetropia.

While, if taken alone, hyperæmia or anæmia of the retina is not to be relied upon as evidence of a similar condition of the brain, if found associated with other symptoms, it may be of considerable value.

Anæmia of the brain is often accompanied by inactivity of the pupillary reflex with dilatation, while contraction of the pupils is characteristic of cerebral congestion.

Cerebral Hemorrhage. In estimating the relative importance and significance of the various ocular manifestations of cerebral hemorrhage or apoplexy, the complex nature of the conditions with which we are dealing should constantly be kept in mind. The location of the hemorrhage, the extent of the extravasation, the suddenness with which it makes its appearance, and the time that has elapsed since its occurrence should all be taken into account, and we must remember that some of the eye symptoms may be due to absolute destruction of certain portions of the brain tissue, while others are the result of paralysis due to temporary pressure in a zone surrounding the clot, and others again are to be attributed to the irritation which occurs in a zone still farther removed from the seat of actual hemorrhage.

A sudden and overwhelming hemorrhage may at first abolish the function of both hemispheres, whereas the later developments, if death does not ensue, will show on which side the lesion is to be found, and for a short time we may have homonymous hemianopsia in the visual field on the side opposite the hemorrhage. Conjugate deviation of the head and eyes toward the side of the lesion is also of frequent occurrence. This is attributed by Knies to irritation occurring in the opposite hemisphere.

Hemorrhage into the visual cortex may in some instances cause very few of the symptoms usually associated with apoplexy. There may be only temporary vertigo with a sudden attack of homonymous hemianopsia. If the hemianopsia is permanent, we may infer that the hemorrhage has been of such a character as to destroy the entire visual centre.

Should the hemorrhage be not too extensive, a fairly accurate diagnosis may sometimes be made at the beginning of an attack, before the more or less confusing secondary and remote symptoms have developed, or after these symptoms have run their course; but during their presence it is difficult accurately to differentiate them from the symptoms which are the essential and permanent results of the lesion.

Hemorrhage into the subarachnoid or subdural space is apt to produce symptoms which resemble those of meningitis. Mydriasis, occasionally as the result of irritation of the sympathetic, but generally due to pressure upon and paralysis of the motor oculi, is not infrequently seen. Myosis is of rare occurrence; it results from hemorrhage into the ventricles, and in some instances, probably from irritation produced by hemorrhage, causing pressure upon the nuclei of the ocular muscles. If a hemorrhage is of such extent as to destroy the primary optic ganglia, the chiasm, or optic tracts, we may have partial or complete atrophy of the optic nerves.

Optic neuritis, choked disk, and partial or complete atrophy of the optic nerves are seen occasionally in cerebral hemorrhage; but evidence supplied by the ophthalmoscope is of far less value in this disease than in the case of tumors or meningitis.

It should be remembered that diseases such as albuminuria, diabetes, atheroma of the bloodvessels, etc., which may predispose to cerebral hemorrhage often produce hemorrhages and other characteristic lesions of the retina and optic nerve, independently of those which may result from extravasations into the tissues of the brain, and in old people the condition of the bloodvessels which favors extravasation into the brain is indicated not infrequently by recurring attacks of conjunctival hemorrhage.

Embolism and Thrombosis of the Cerebral Vessels. A cerebral embolism, if not infectious, leads to degeneration or softening and necrosis of the brain tissue supplied by the vessel whose course it obstructs, and a non-infectious thrombosis or a circumscribed hemorrhage will produce similar effects, though the clear-cut and well-defined lesion produced by an embolus often renders it possible to determine quite accurately its location by the resulting focal symptoms; and if it is situated in the visual cortex, in the occipital lobe, in the primary optic ganglia, or in the course of the optic tracts, in the cortical centres or primary ganglia of the motor nerves of the eye, the characteristic paralytic symptoms will develop promptly after a brief period of reaction. Symmetrical, bilateral softening of the optic centres has been reported in a number of instances, and Wilbrand mentions one case in which double choked disk occurred (Knies).

An infectious embolus or thrombosis leads to the formation of a cerebral abscess with its characteristic symptoms, while an infectious thrombosis of the cavernous sinus may, in addition to the other symptoms of an abscess at the base of the brain, result in orbital suppuration with its long train of disastrous effects. The eyeball is protruded and fixed by infiltration of the orbital tissues, the conjunctiva becomes chemotic, the lids swollen, and there follows blindness with a widely dilated and immovable pupil. If the thrombotic process includes the ophthalmic vein, the ophthalmoscope reveals distended retinal veins, with injection of the nerve head and retinal hemorrhages. Later, as the infectious process extends, we have opacity and ulceration of the cornea, and finally panophthalmitis.

In the early stages of the infectious process, and during the progress of a non-infective or marantic thrombosis of the cavernous sinus, the ophthalmoscope renders most valuable assistance, especially in distinguishing the latter condition from meningitis. In both meningitis and non-infectious thrombosis of the cavernous sinus there may be paralysis of the motor nerves, insensibility of the trigeminus, with, its consequences in conjunctival and corneal anæsthesia, partial or complete paralysis of the optic nerve, and more or less œdema of the lids, and protrusion of the eyeball from involvement of the orbit. In meningitis we may have congestion and even pronounced optic neuritis; "but the marked stasis of the retinal veins which is found in thrombosis of the sinus with thrombosis of the ophthalmic vein is never observed" (Knies).

Abscess of the Brain. The ocular symptoms produced by abscess of the brain may be the same as those about to be enumerated as resulting from tumor; but, in addition, there is the general evidence of an infective process acting as a cause; and it should be borne in mind that some of the ocular manifestations, such as septic choroiditis, embolism, and thrombosis, may result directly from this infective process rather than from the coincident cerebral abscess.

As in tumor, we may find diffuse and local effects manifested in choked disk and obstructive neuritis, which latter is apt to be bilateral, and is the most characteristic ophthalmoscopic manifestation. There may be paralysis of the motor and sensory nerves, preceded by spastic contractions of muscles and other evidences of the existence of a zone of irritation near the abscess. As the abscess may be practically stationary or rapidly progressive, the various ocular manifestations may be of prolonged duration or follow one another in quick succession, and in the event of a rupture we may have a fatal termination preceded by the ocular and general evidences of purulent meningitis, with paralysis of the fifth nerve, neuroparalytic keratitis, etc. Perforation into the ventricles may be attended by marked myosis, which is attributed by Knies to direct irritation of the sphincter nuclei.

The prognosis after operation, so far as the eye is concerned, depends upon the location and extent of the damage to the brain tissue. The irritative symptoms may subside, as may the optic neuritis, and to some extent vision may be restored; but if the visual cortex has been seriously involved, we are apt to have not only impaired vision and limitation of the visual fields, but also defective color sense (Knies).

Tumors of the Brain. Almost all forms of neoplasm are found in the cranial cavity, although some, such as lipoma, which are common in other localities, very rarely appear in the brain. Tubercular tumors are more common in the brain than elsewhere, and sarcomata and syphilitic tumors are of frequent occurrence, the latter being often associated with gummatous meningitis. Glioma is almost exclusively a cerebral tumor, being found in the brain and spinal cord, and in no other part of the body, excepting the retina, from which it often extends to the brain.

The symptoms produced in the eye, as in other parts of the peripheral nervous system, differ greatly in accordance with the size, period of growth, and location of the tumor; and it sometimes happens that a growth which later causes irritation, and finally destruction of the parts with which it lies in contact, may in the earlier stages of its development produce only general symptoms of diffuse intracranial pressure. Tumors of the anterior and middle fossa may invade the orbit and cause exophthalmos.

Choked disk or optic neuritis, usually double, and attacks of temporary total loss of sight due to general intracranial pressure, indicate the presence of a tumor of the brain, although other evidence is neces-

sary to enable us to form an opinion as to its location. The size of a tumor seems to have little influence, and the choked disk or optic neuritis which appears is not merely the result of mechanical pressure.

In addition to the general symptoms of cerebral compression accompanying brain tumor, such as headache, hebetude, drowsiness, vomiting, a slow pulse, and dilatation of the pupils, there is sometimes evidence of pressure on parts far removed from the seat of the growth, which is apt to be very misleading in our attempts at localization. Owing to its long course, the sixth nerve is especially apt to be affected by such indirect pressure, producing paralysis of the external rectus.

If the tumor is of such a nature as to provoke irritation as well as pressure, we may have conjugate deviation of the eyes and head, concentric narrowing of the visual fields, and paroxysmal attacks of bilateral blindness.

While choked disk is not always one of the early symptoms of brain tumor, it is one of the most important, appearing at some stage in about 80 per cent. of the cases; and if this symptom is not found in some stage, the other evidence on which the diagnosis of cerebral neoplasm is based should be very convincing. It is said to be more frequent in tumors of the cerebellum than in those of the frontal lobes, and may be produced by a tumor in any part of the brain, although, if the neoplasm is in the membranes on the convexity and merely compresses the brain, it is less apt to produce choked disk or optic neuritis than when it invades the cerebral tissues (Gowers). Choked disk is sometimes found even in tumor of the spinal cord. Some eminent writers on nervous diseases ignore the distinction between choked disk and obstructive neuritis, but it is, nevertheless, an important one, for, while undoubtedly some degree of neuritis finally develops in almost every case of choked disk, there often exists in the early stages a true œdema of the nerve head in which the tissues are quite translucent and do not present the appearance of vascularity and inflammation seen in optic neuritis. This œdema is not necessarily accompanied by marked impairment of vision. Choked disk with tumor of the brain is usually, although not always, bilateral, and it does not necessarily indicate that the growth is large or is located near those parts of the brain which are especially concerned with vision. Indeed, while rapidly growing and large tumors are apt to produce it, small growths by causing, as they often do, dropsy of the ventricles, are accompanied not infrequently by marked œdema of the nerve head.

Choked disk and optic neuritis, if maintained for a sufficient length of time, will almost inevitably lead to contraction of the visual field, with extension of the blind spot, and sometimes central scotoma, followed by atrophy of the optic nerve and blindness. Exceptions to this are found in those rare cases in which a cure is effected by operative or other measures, and especially in neoplasms of syphilitic origin, when in the proper stage active treatment with mercurials and iodides succeeds in bringing about absorption.

In a number of instances[1] where operative interference has been unsuccessful so far as removal of the tumor was concerned, marked freedom from pain and restoration of vision have followed the relief from pressure on the cerebral tissue.

While choked disk is one of the most positive indications of the presence of an intracranial growth, it should be borne in mind that it is simply an œdema of the nerve head accompanied by distention of the sheath of the nerve, and that there are other conditions as well as tumor which may produce it occasionally. Among these may be mentioned abscess of the brain and cerebral hemorrhage. It has also been seen after profuse hemorrhage in other portions of the body and in cases of leukæmia, albuminuria, and diabetes; but there is usually other evidence to aid in establishing the diagnosis.

Tumors of the brain in a certain proportion of cases cause optic neuritis, followed by atrophy without choked disk. "Oppenheim observed typical choked disk fourteen times, neuritis five times, and hyperæmia of the papilla once" (Knies). And we may in some cases have atrophy of the nerve without either choked disk or neuritis.

Taken with other evidence, choked disk, obstructive neuritis, simple optic neuritis, and progressive atrophy of the optic nerve are of great value in determining the character of a brain lesion; but alone they should not be considered as a sufficient basis on which to make a positive diagnosis.

Aneurism, by pressure and irritation, produces effects upon the eyes similar to those resulting from other tumors, and in very rare and exceptional instances a tumor may be so situated as to produce a group of focal eye symptoms almost as clearly defined as those sometimes observed in cases of embolism and softening. These symptoms may present themselves in the form of cortical blindness or hemianopsia, mind blindness, alexia, visual aphasia, dyslexia, amnesic color blindness, and visual hallucinations, or cortical disturbance of the ocular movements, such as conjugate deviation of the eyes, often accompanied by deviation of the head in the same direction. If the tumor happens to press upon the gray matter around the aqueduct of Sylvius or in the floor of the fourth ventricle, it produces nuclear ocular palsy or ophthalmoplegia—external if affecting the orbital muscles, as the recti and oblique muscles; or internal if affecting the iris and ciliary muscle. If the tumor lies in such a position as to affect the efferent fibres of the ocular nerves in the crus cerebri or pons, between the nuclei and their point of emergence at the base of the brain, we have what is termed *fascicular paralysis* of the third, fifth, sixth, or seventh nerve, which is sometimes spoken of as crossed or alternate paralysis. And when situated at the base, in addition to its effect upon the optic tract, a tumor may cause paralysis of any or all of the nerves supplying the external and internal ocular muscles, as well as the fifth nerve.

[1] Swanzy, in Norris and Oliver's System of Diseases of the Eye, vol. iv, p. 545.

If found in the corpora quadrigemina, a tumor may produce oculomotor paralysis, a reeling gait, with possibly blindness and deafness.

The differential diagnosis between tumor and abscess of the brain is not always easily made. They may have in common headache, vomiting, choked disk, or optic neuritis (generally double), and mental disturbance; while tumor is apt to cause in addition the well-marked focal symptoms enumerated above, with, at times, hemiplegia. Fever and rigor favor abscess. The cause of abscess is often very clear, being frequently traceable to a focus of suppuration, such as purulent otitis media, while that of tumor is obscure.

Meningitis in its various forms gives rise to a variety of ocular lesions of the most serious character. In general they are the direct result of the action of the exudate upon the visual centres, ganglia, or optic tracts, and upon the points of origin or trunks of the motor and sensory nerves; or they may be due to secondary infection of the eye from the septic material which is characteristic of the meningeal inflammation. If the process is extensive, we may find the eye congested, hyperæsthetic, and sensitive to light in the early stages; and soon symptoms will develop which indicate whether the inflammatory process affects the convexity of the brain or the base. If the former, we may have in the early stages an homonymous hemianopsia with the pupillary reaction to light preserved, or both cortical centres may be involved, affecting both halves of the retina of each eye.

In acute cases the inflammatory process usually extends rapidly, so that conjugate deviation and other symptoms pointing to a cortical lesion are transitory in character; and as in a large proportion of cases of meningitis there is an exudate at the base of the brain, the ocular manifestations are apt to be peripheral and the result of either irritation or paralysis of the nerve trunks which are embedded in the exudate and reach the eye through the apex of the orbit.

The abducens is attacked most frequently, the motor oculi rarely; and the presence of hyperæsthesia, paræsthesia, and anæsthesia in the cutaneous surface of the face, with neuroparalytic keratitis, indicates involvement of the trigeminus.

Among the symptoms of irritation we may have contraction of the ocular muscles, producing various forms of strabismus, and rarely nystagmus.

Paralysis of the facial nerve, leading to lagophthalmos, which may be accompanied by deafness from involvement of the auditory nerve, is one of the possible results when the exudate is found in the middle fossa. In basilar meningitis vision may be affected by involvement of the tractus, by optic neuritis, extension to the orbits, generally along the veins, producing chemosis, and perhaps later orbital cellulitis, fixation, and protrusion of the eyeballs, etc., or by the production of a seroplastic or purulent choroiditis sometimes, though rarely, terminating in panophthalmitis.

A degree of optic neuritis presents itself in some stage of the majority of cases of cerebral meningitis, and although, unfortunately,

it sometimes happens that it cannot be detected sufficiently early to be the means of establishing the diagnosis, in many instances it is of the utmost value. When fully developed, it is generally bilateral.

Doubt as to the differential diagnosis between typhoid fever or pneumonia on the one hand, and meningitis on the other, may sometimes be decided by means of the ophthalmoscope.

Optic neuritis, as seen with the ophthalmoscope, may vary in degree from simple hyperæmia to a decided papillitis, although great swelling of the disk is not often seen, as in cerebral tumor and some cases of abscess. There is usually an absence of pronounced exudation and hemorrhage; but a certain cloudiness of the tissues of the nerve head, with blurring and indistinctness of its outlines, is in keeping with the fact that the microscope reveals infiltration of the pial sheath and connective tissue, especially toward the periphery (Knies).

A dense, chalky-white disk with sharp outlines and marked narrowing of the bloodvessels, is seen in the atrophic stage, with often complete blindness, though in some cases the amount of vision remaining seems entirely out of proportion to the evidences of atrophy as seen with the ophthalmoscope. When some vision remains, however, we are apt to have irregular narrowing of the visual fields, scotomata, and defective color sense.

A septic ("metastatic") exudative choroiditis sometimes develops in the early stages of simple meningitis, especially in young children, although it may also appear at a late period and in other forms of the disease; or it may be discovered after the active symptoms have subsided. It is generally unilateral, although both eyes may be affected; and it is said to be of embolic origin, sometimes being found in cases of ulcerative endocarditis, in puerperal fever, recurrent fever, typhoid fever, scarlatina, mumps, erysipelas, etc. (Noyes).

This condition of the eye not infrequently escapes the attention of the attending physician during the active period of the disease to which it owes its origin, but usually presents well-marked and easily discoverable local symptoms of a low grade of irido-cyclo-choroiditis, sometimes accompanied by marked ciliary injection, iritic exudation and adhesions, parenchymatous keratitis, and even hypopyon. If the above symptoms of iritis and keratitis are absent, the ophthalmoscope, or sometimes oblique illumination, will reveal the purulent exudate in the choroid and retina, which often extends so far forward in the vitreous chamber as to lie in contact with the posterior surface of the lens, and, especially when bloodvessels develop on its surface, it closely resembles glioma of the retina—*pseudoglioma*. Such eyes are usually soft and easily irritated, but sometimes retain their normal external appearance, although often in later years they undergo degenerative changes, becoming quadrate under the pressure of the recti muscles, and developing degenerative keratitis and calcification of the crystalline lens.

Instances have been recorded in which, after the formation of a moderate amount of exudate, absorption has taken place and vision

has been restored, though such cases must be extremely rare. We may also have in meningitis, as in certain other diseases accompanied by typhoid symptoms, mild grades of cyclitis and choroiditis, which sometimes recover without leaving serious impairment of vision.

Acute tubercular meningitis is in about 15 per cent. of the cases accompanied by miliary tubercles in the choroid, which may be made out with the ophthalmoscope as pale yellowish spots which are somewhat prominent and vary in size from 0.5 mm. to 2.5 mm. They are unaccompanied by pigmentation, and seem to be more common in the neighborhood of the macula lutea and disk. More are often found post-mortem.

Paralyses of the ocular muscles are often seen in tubercular meningitis, as it is prone to attack the base of the brain, and optic neuritis is more common in this than in any other form, especially if the tubercular exudate at any point appears as a tumor.

Cerebro-spinal meningitis is very apt at some stage to be the cause of most serious involvement of the eyes. In the early stages we may have swelling of the lids, conjunctivitis with œdema, and photophobia with contracted or dilated pupils which are often unequal. Keratitis is not uncommon, and iridochoroiditis and retinitis with optic neuritis, or paralysis of the optic nerve without apparent neuritis, are of frequent occurrence. What has been said under the head of septic or metastatic choroiditis as occurring in meningitis in general, applies especially to this form of the disease.

Whether or not the pneumococci reach the eye through the lymph spaces of the optic nerve has not as yet been proved (Axenfeld); but that, in some instances, they reach it by way of the circulation through general systemic embolic poisoning has been established.[1]

The prognosis as to life, and especially as to sight, is most grave.

Pachymeningitis produces eye symptoms which vary with its location. As it is most frequently found on the convexity of the brain, the eye symptoms are apt to be cortical in nature, though when associated with hemorrhage the more diffuse symptoms usually seen with the tumor may be added.

A circumscribed meningitis presenting few of the other symptoms found with more general inflammation of the membranes may be accompanied in the early stages by color phantasms, nyctalopia, etc., and at a later period cause impaired vision, scotomata, limitation of the visual fields, and disturbance of color sense. The ophthalmoscope may at first reveal neuritis, which is followed by more or less complete atrophy of the optic nerve (Knies).

Metastatic purulent meningitis may result from purulent inflammation of the eye, especially from traumatic panophthalmitis.

A number of instances have been recorded, some of which have occurred after enucleation, and this has been used as an argument against enucleation in panophthalmitis; but both logic and expe-

[1] Swanzy, in Norris and Oliver's System of Diseases of the Eye.

rience teach us that the case must be an unusual one, indeed, in which removal of such a source of infection will not increase the patient's chance of escaping meningitis.

Insanity. While the insomnia and excitement incident to many forms of insanity may lead to marked injection of the bulbar conjunctiva, and while variations in the pupil may be noted, and atrophic and degenerative disease of the nerve, retina, and choroid be found as the result of a disease which is a direct or indirect cause of mental disorders, it cannot properly be said that there is any affection of the eye which can be directly attributed to insanity.

Hallucinations of sight which are visual perceptions not founded on an objective reality, and visual illusions which are misinterpretations of sensory images, when they cease to be recognized by the subject as hallucinations and illusions, are among the more common manifestations of insanity.

Post-operative delirium, and even insanity are by no means uncommon after iridectomy and the extraction of cataract; and when we consider the prolonged suspense, the state of mental excitement with physical inactivity incident to the operation and after-treatment, and the fact that all light is generally excluded, it is not strange that judgment sometimes ceases to hold sway over the hallucinations excited by such an ordeal. It is a fact, however, that such mental disturbances seldom manifest themselves excepting among those predisposed to such affections.

It sometimes happens after operation upon the eye that the tendency to delirium is greatly increased by the effect of atropine, used to prevent the formation of iritic adhesions, and caution in its administration may prevent the development of most troublesome symptoms.

General Paralysis of the Insane. In view of the wide distribution and character of the cerebral lesions in paretic dementia, it is not strange that we should have a variety of eye symptoms which, owing to the fact that they often make their appearance at an early period, are of the greatest diagnostic and prognostic value.

Trophic and vasomotor disorders occur in the eye as elsewhere; but it is to the cortical visual disturbances, mind blindness, and hemianopsia, paroxysmal or permanent, and to atrophy of the optic nerve, and especially disturbances of innervation of the intrinsic and extrinsic ocular muscles, that our attention will be directed.

Paralysis of the orbital muscles, cycloplegia, and pupillary anomalies, such as mydriasis, myosis, irregularity of shape, inequality in the two eyes, and disturbance of the pupillary reflex often appear in the prodromal stage; but, as a great variety of cerebral lesions due to widely different causes may produce similar symptoms, the largest experience and the utmost caution and judgment are often necessary to enable the observer to interpret them correctly. Their value should carefully be estimated when taken in connection with the evidence derived from other sources.

Many striking cases are on record in which some comparatively slight pupillary anomaly has served as the warning note of approaching insanity; but every ophthalmologist of wide experience sees numerous cases of pupillary anomalies and unaccountable paralysis of the extrinsic ocular muscles which are never followed by such dire consequences; and while these symptoms are undoubtedly of great significance, the necessarily complicated nature of the subject and the limitations of our knowledge of the brain should warn us to exercise caution in our attempts to interpret them.

Mind blindness when present in dementia is generally, though not always, found in the later stages. Schweigger reports a most remarkable case treated by Wernicke in which, "with good acuteness of vision and without any absolute defect in the field, there were distributed over a great portion of the field a number of relative scotomata, within the area of any one of which, although objects could be seen by the patient, yet he could not tell what they were."[1]

Mind blindness may be paroxysmal, continuing for several days and then disappearing. Though it is always temporary, it is apt to be followed by actual blindness as the disease progresses. Hallucinations of sight, in some cases unilateral, are very common, and sometimes appear as early symptoms.

Atrophy of the optic nerve may occur in the early stages or even precede mental disturbance, but is usually a late symptom. It appears merely as an incident in the course of the organic cerebral lesions, of which the general paralysis and insanity are symptoms, and is not of very frequent occurrence, being found, according to Gudden, in about 4.9 per cent. of a series of 1386 cases.

Hyperæmia of the papilla, and even a slight degree of optic neuritis, have been observed in a very small percentage of cases.

Pupillary anomalies and disturbances of the ciliary and orbital muscles are the most significant ocular symptoms in general paralysis of the insane. The pupils are usually contracted in the early stages, although later they are often more or less dilated; but what is termed reflex rigidity of the pupils, in which response to light stimulus may be diminished or absent, and later reaction to convergence and accommodation may fail, or in which the pupils are equal, or one or both assume an irregular shape, is one of the most valuable of the early symptoms. Among 500 cases Moeli found reflex rigidity present in 47 per cent., doubtful reaction in 4 per cent., and sluggish reaction in 10 per cent.; and among 205 patients with reflex pupillary rigidity Thomsen found 83 per cent. of general paresis (Knies). It should be remembered, however, that although other diseases rarely produce this symptom, absence of pupillary reaction to light and reflex rigidity of the pupil are among the more common ear symptoms of tabes dorsalis, as well as of general paralysis of the insane.

The study of pupillary reactions in nervous diseases is necessarily

[1] Swanzy, in Norris and Oliver's System of Diseases of the Eye.

intricate and involved, but it has by some writers been rendered still more complicated by giving minute attention to unimportant details.

Paralysis of accommodation is of far less frequent occurrence than pupillary anomalies, being found by Moeli in about 1.5 per cent. of all cases (Knies).

Although not so common a symptom as mydriasis, nuclear paralysis or paralysis of the orbital muscles is occasionally seen. It may result in loss of power in the third, fourth, or more frequently the sixth nerve, with the accompanying diplopia and strabismus or ptosis. While usually temporary, it is not always so, and is prone to relapse. According to Schütz, Siemering, and Boediker, the above oculomotor paralyses are "caused by degenerative changes in the central gray matter of the aqueduct of Sylvius and fourth ventricle."[1]

Ptosis, twitching of the eyelids, and transient nystagmus may all be found in a limited number of cases, and among other motor disturbances of cortical origin we not infrequently have conjugate deviation of the head and eyes.

Several writers have mentioned ocular migraine or scintillating scotoma as a not infrequent premonitory symptom of paretic dementia, but this is of such frequent occurrence in other conditions that it is certainly not a symptom of great diagnostic value.

Diffuse cerebral sclerosis is apt to be accompanied by impaired pupillary reaction, and cases of paralysis of the sixth nerve and nystagmus, as well as optic neuritis, have been reported.

In **paralysis agitans,** or **Wilkinson's disease,** a bilateral or rarely a unilateral tremor may sometimes be noticed in the muscles of the margin of the upper lid. This is more marked when the lids are closed, and is accompanied by a degree of rigidity on attempting to open them. Nystagmus is a rare symptom.

According to Gowers, the slowness of motion which is noticeable in other portions of the muscular system rarely affects the orbital muscles. The patient will turn the eyes instantly in any desired direction, and follow them slowly with the head by the action of the muscles of the neck.

Spasm of accommodation has been noted in several cases by Koenig, and gray atrophy and bilateral ptosis have occasionally been reported.

Disseminated sclerosis in a large proportion of cases is accompanied by very significant and characteristic eye symptoms which may be of great diagnostic value. They manifest themselves in defective vision, a variety of forms of limitation of the visual and color fields, color, and, in rare instances, absolute scotomata, variations in the ophthalmoscopic appearance of the disk, and disturbances of the ocular and orbital muscles. The onset of these symptoms may be gradual, but more often they come on suddenly. They may affect one or both eyes, and they vary in degree, sometimes disappearing entirely, and in other instances relapsing after an interval of many

[1] Swanzy, in Norris and Oliver's System of Diseases of the Eye.

weeks. Amaurosis, which is rarely complete and permanent, may continue for several months, and, after prolonged remission, appear again (Charcot). It is apt to be accompanied by the sensation of a mist before the eyes, and even when scotomata are present these are rarely absolute. Pure cases of disseminated sclerosis are not accompanied by hemianopsia, and this tends to prove that the lesions, like those of retrobulbar neuritis, are not in the chiasm or optic tracts, but in the optic nerve itself.

Visual defects and changes in the appearance of the optic disk may precede the other symptoms of disseminated sclerosis by months or years, or they may make their appearance in the early stages; but they are usually found only after the general symptoms are well developed.[1]

Among the most striking ocular manifestations of disseminated sclerosis are the disorders of the ocular and orbital muscles, and of these the most important are the nystagmus, ataxic nystagmic twitchings, and tremors attributed by Knies to insufficient cortical innervation of the nuclei, which he believes to be due in the main to perinuclear foci in the fibres of the corona radiata.

Nystagmus, which is very rare in other forms of nervous disease, is said to occur in about one-half of the cases of disseminated sclerosis.

Either with or without nystagmus we may have disorders of the associated movements of the eyes. This was observed by Uthoff in three out of 100 cases. Isolated paralysis of the external ocular muscles and nuclear paralysis may occur, an example of the latter being defective conjugate motion to the right or left and paresis of the power of convergence (Swanzy). The peripheral nerves have been found in a number of instances to be the seat of sclerotic foci, Uthoff having noted four cases of unilateral abducens paralysis and three cases of partial paralysis of the motor oculi. Moderate degrees of impairment of motility are very common. As in the case of paralysis of the optic nerve, the motor affections are much more frequently seen after the other symptoms are well developed, but they may occur in the early stages, or even before any other evidences of the disease have appeared.

Abnormal pupillary reaction is uncommon in disseminated sclerosis, but a few cases have been observed including reflex rigidity of the pupils, myosis in the advanced stages, impaired light and convergence reaction, inequality of size, and hippus. Uthoff found slight deviations from the normal in 16 per cent. of the cases studied by him.

As Swanzy has pointed out, the fact that ophthalmoscopic changes in the disk may be observed in about 50 per cent. of the cases is of value in establishing the diagnosis between disseminated sclerosis and hysteria, in which we sometimes find symptoms resembling those of the earlier stage of the former disease. And we are also assisted

[1] Swanzy, in Norris and Oliver's System of Diseases of the Eye.

by the rarity of central scotoma and the irregular and erratic character of the visual and color fields in hysteria, as compared with those of disseminated sclerosis which, although narrow, follow the regular physiological order.

In amaurosis, scotomata, and impairment of the visual fields the symptoms indicate retrobulbar disease of the optic nerve, and granular degeneration of the medullary sheaths, with intact axis-cylinders, may be found with other evidence of interstitial neuritis. Degenerative foci are undoubtedly sometimes found in the primary optic ganglion, and rarely in the chiasm and tractus, but they are of far more frequent occurrence in the optic nerves.

According to Knies, the pathological process in the optic nerve "stands midway between pronounced optic neuritis and simple atrophy."

While the medullary sheaths are destroyed, a large proportion of the axis-cylinders escape, although from time to time in the progress of the disease their conductivity may be impaired. This accounts for the fact that the disturbance of vision or the defect in the visual field may be very pronounced, while the optic disk appears normal, and that vision may vary from time to time, or be in a measure restored so long as the axis-cylinders which pass through the sclerosed patches are not actually destroyed. Optic neuritis, according to Uthoff, is found in about 5 per cent. of the cases, but the ophthalmoscope reveals no retinal atrophy, and there may be no visible evidence whatever of a defect of the optic nerve. Decided atrophy occurs in only about 3 per cent. of the cases, and even partial atrophy in only 19 per cent. In a considerable number of cases (about 18 per cent.) an ophthalmoscopic picture is observed closely resembling that of toxic amblyopia. This, however, is not in every case accompanied by the characteristic central scotoma.

When the ntrinsic and extrinsic ocular muscles are affected, the lesions are in the main nuclear, although in a number of instances sclerotic foci have been found in the peripheral nerves. Leube saw both motor oculi nerves converted into thick gray bands (Knies).

Deformities of the skull following chronic meningitis in infants, producing premature ossification of the cranial bones and narrowing of the optic foramina, result first in optic neuritis and later in atrophy of the optic nerve.

Hydrocephalus as seen in infancy may be accompanied by optic neuritis or atrophy of the optic nerves, but this is not of frequent occurrence; while hydrocephalus appearing later in life, when the sutures are more firmly united, is as a rule accompanied by such symptoms and by evidences of pressure closely resembling those of tumor of the brain.

Swanzy, in Norris and Oliver's *System of Diseases of the Eye*, calls attention to the occurrence of bitemporal hemianopsia in hydrocephalus, due to pressure on the optic commissure by the distended floor of the third ventricle.

In the various forms of meningitis, as well as in hydrocephalus, there is often such a lowered state of vitality as to lead to the development of keratitis and conjunctivitis.

Porencephalus (cysts or cavities in the cortex) may be accompanied by eye symptoms somewhat similar to those which appear in softening. Nystagmus, ptosis, reflex rigidity of the pupil, and gray atrophy of the optic nerve have all been observed.

Bulbar paralysis in its typical form is not accompanied by eye symptoms, but due, as it is, to pathological processes in the medulla oblongata, it sometimes has associated with it lesions of the visual and especially the motor centres of the eye. Optic nerve atrophy has been observed, and several writers have reported cases of paralysis of the ocular and orbital muscles, with resulting dilatation and fixation of the pupils, nystagmus, ocular deviation or fixation, and even a degree of exophthalmos. Conjugate deviation and paralysis of the ocular branch of the facial have been noted, and while the disease is at its height concentric limitation of the visual field and slight impairment of vision may occur. Straminski, of Wilna, reports a case of this kind following influenza, and terminating in recovery in nine months

Progressive ophthalmoplegia, or what in contradistinction to pure bulbar paralysis has been designated as superior poliencephalitis in its acute form, is due to hemorrhagic inflammation of the gray matter in the floor of the fourth ventricle and the aqueduct of Sylvius (Knies). In addition to the somnolence, which is characteristic, there is progressive paralysis of the ocular muscles, and this may in a short time be quite complete.

In the chronic form there is degeneration, which may affect the nuclei, nerves, or muscles, producing progressive paralyses, which are usually bilateral, although irregular, and may be complete or incomplete. These paralyses, with the resulting convergence, divergence, nystagmus, reflex rigidity of the pupil, etc., may disappear in whole or in part, only to appear again and continue to advance (Knies).

Syringomyelia is accompanied sometimes by concentric contraction of the visual fields and rarely by optic neuritis. Abducens paralysis and nystagmus have also been reported.

DISEASES OF THE SPINAL CORD.

Myelitis is accompanied sometimes by optic neuritis, which may recover or go on to complete blindness; and Swanzy points out that if the cervical portion of the cord is involved we may have mydriasis due to irritation, or paralytic myosis.

Tabes dorsalis is in a large proportion of cases accompanied or preceded by eye symptoms which are of the greatest importance and often supply the first positive indication of the nature of this serious disease. These consist of atrophy of the optic nerve, par-

alysis and ataxy of the ocular muscles, pupillary alterations, and paresis or paralysis of accommodation.

Associated, as tabes often is, with other diffuse cerebral and spinal disease, the ocular symptoms afford important assistance in establishing the diagnosis. Atrophy of the optic nerve is found in about 20 per cent. of the cases, and, when fully developed, the disk is gray, is often slightly cupped, revealing the delicate fibres of the lamina cribrosa, and, especially in cases with a deep physiological depression, is difficult to distinguish from glaucoma simplex.

In the earlier stages the grayish discoloration of the disk is more noticeable on the temporal side than on the nasal, which is normally more pink than the outer half, but as the disease advances the whole surface becomes uniformly gray, and the arteries and veins are reduced to narrow threads.

Whatever may be the cause of tabes, it is evident that the atrophic process in the optic nerve is not a direct extension of the disease in the brain and cord, but a separate manifestation of the same process. The fibres in the centre of the nerve are affected last, and Leber has shown that it begins in the periphery of the retrobulbar portion and extends toward the axis. The gray atrophy may also sometimes be detected in the chiasm, the tractus, and even as far as the primary optic ganglia (Knies).

Some writers claim that in the very early stages hyperæmia of the disk precedes atrophy of the optic nerve, but this is not established, and on theoretical grounds it would seem improbable, as the process is a primary atrophy of the nervous elements. Both histologically and with the ophthalmoscope, tabetic atrophy is easily distinguished from postneuritic white atrophy, in which a dense white or yellowish-white disk is seen entirely obscuring the fibres of the lamina cribrosa. Atrophy of the optic nerve, while sometimes appearing in the later stages of locomotor ataxia, may antedate the appearance of ataxia or even precede the lightning pains, loss of knee-jerk, and other spinal symptoms from two to twenty years.[1]

Ophthalmoscopic evidence of atrophy generally precedes disturbance of vision, but the visual disturbance may in some cases be much more marked than would be indicated by the appearance of the disk.

Strange as it may seem, many writers maintain that the progress of the general disease is checked and there is an abatement of the spinal symptoms on the development of optic atrophy if it appears in the preataxic stage.[2] In regard to this, Knies very properly remarks that a mistaken diagnosis may account for the improvement, as the prognosis is often much more favorable in some of the neurotic and sclerotic processes which may be mistaken for tabes.

Authorities differ greatly as to the frequency of the occurrence of optic atrophy in tabes, Gowers placing it at 13.5 per cent., while

[1] Swanzy, in Norris and Oliver's System of Diseases of the Eye.
[2] Benedict, Wien. med. Presse, 1881, Nos. 1, 2, 3, 4, 5.

Berger gives 33.7 per cent., and Uthoff 20 per cent. Although both eyes usually are affected at the same time, or with a very short interval, years sometimes elapse after one nerve becomes atrophic before the other is involved.

Galezowski has estimated that two-thirds of all optic nerve atrophies are of tabetic origin; but whether this be correct or not, Knies states that genuine gray atrophy of the optic nerve should always lead to the suspicion of tabes, as even ten to thirty years may elapse after the appearance of gray atrophy of the optic nerve before the other symptoms develop.

Swanzy calls attention to the fact, which is omitted by some writers, that at the beginning we may have such subjective phenomena as photophobia, a sensation of sparks and colored lights, and that the patients complain of a fog or smoke before the eyes. He also points out that the interval between the appearance of optic nerve disease and the development of complete blindness may vary from a few months to seventeen years. One year would represent a probable average. A temporary or permanent arrest of progress may in some instances be observed, but this is of rare occurrence in spite of the most skilful and persistent treatment.

Martin calls attention to the fact that tabetic patients when blind do not generally sway from side to side in closing the eyes and standing with the feet together, a very common symptom in those who are still able to see.

The optic atrophy of tabes is accompanied by failure of central and color vision and narrowing of the visual and color fields; and with diminishing illumination the vision of a tabetic patient is often found to fail much more rapidly in proportion to his usual standard than is the case with one with normal optic nerves. The contraction of the visual fields is generally concentric to the blind spot; but there are many exceptions to this rule. The defects vary greatly in their outlines, and no one form or mode of development can, as in the case of glaucoma, be said to be characteristic. Even when it finally becomes concentric, narrowing of the field often begins by the formation of a re-entering angle of blindness, and, as the disease progresses, a small eccentric portion of the retina sometimes remains intact after the macular region is blind.

Central scotomata, if they occur at all, are very rare, and should, as Knies has pointed out, arouse the suspicion of a complication or a mistaken diagnosis. Central vision may in some cases remain fairly good in spite of narrowing of the visual field, or, on the other hand, we may have marked impairment of central vision with a normal peripheral field; but usually impairment of the visual field and loss of central vision as they progress bear a definite relation to one another. Swanzy refers to the fact that a temporary functional increase in the narrowing of the visual fields may result from mental worry.

Color blindness is almost always found in association with the optic atrophy of tabes, but its degree is not necessarily in direct

proportion to the visual disturbance nor to the limitation of the visual field. In some instances it may even be detected before optic atrophy can be discovered with the ophthalmoscope.

An examination of the color fields is very important, as those cases in which their narrowing is much more marked than the field for white are apt to be rapidly progressive; and Knies has pointed out that a re-entering angle in the boundary of the color field is the forerunner of a similar narrowing of the field for white.

As rare visual disorders, we may in the later stages have symmetrical defects of the visual fields or homonymous hemianopsia.

Disorders of the ocular muscles are of frequent occurrence in tabes, and their importance is greatly increased by the fact that in a large proportion of cases they appear in the early stages, mydriasis, diplopia, or ptosis often being the first symptom to attract the attention of the patient. According to Uthoff, they are found in 20 per cent., and according to Berger in 38 per cent. of all cases. They usually develop suddenly, and while they may be permanent, more frequently disappear after a period varying from a few hours to a year or more; but they are prone to relapse, and they are more apt to be permanent if occurring in the later stages of the disease. The sudden appearance of paralysis of an ocular muscle in an apparently healthy person, especially if it recovers in a short time or subsequently relapses, should always arouse the suspicion of tabes (Knies).

Any one or more of the orbital nerves may be affected, but paralysis and paresis of the abducens and motor oculi are of most frequent occurrence.

The lesion in paralysis of tabetic origin, while it may be perinuclear or internuclear, is generally peripheral or nuclear, and therefore we never have associated or conjugate paralysis (Knies). While true nystagmus is of very rare occurrence in tabes, we not infrequently may observe ataxic oscillations or what are sometimes termed nystagmic twitchings, which may be demonstrated by causing the eye to follow an object in motion and fix it when the motion ceases.

Paralysis of the ocular branch of the facial nerve is of rare occurrence, but paresis accompanied by tremor on effort to close the lid is frequently observed.

Pupillary alterations are of the greatest diagnostic importance in tabes. The pupils may be of unequal size in the two eyes, and are very often of irregular shape. Mydriasis, due to oculomotor paralysis, but not necessarily accompanied by cycloplegia, is sometimes observed, though it is rare. Myosis, on the other hand, is very common, and may be looked upon as characteristic of tabes dorsalis. The contraction may be extreme ("pin-hole pupil") or of only moderate degree; but whether normal in size or contracted, our attention in this disease is especially directed to the absence of reaction to light, accommodative effort, convergence, and cutaneous irritation, which appear in the various stages as the tabetic changes progress. These pupil changes are due to disease of the ciliospinal centre.

The Argyll-Robertson pupil, while it is also occasionally observed in other nervous diseases, is a most important symptom of tabes dorsalis. It consists in the absence of contraction on exposure to light of a pupil which still retains the power to contract in convergence or accommodation. The pupil may be normal, or even dilated, although it is generally quite narrow, and it should be borne in mind that later, when the disease is sufficiently advanced, and after light stimulus has ceased to cause contraction, the reflex to sensitive stimuli, such as cutaneous irritation, is lost, and finally convergence also fails to be accompanied by contraction.

While occasionally entirely wanting, the Argyll-Robertson pupil is one of the most constant of all the symptoms of tabes, and, owing to the fact that it is often an initial symptom, its value can scarcely be overestimated. Dillman found it in 76 per cent. of his cases. In 31.6 per cent. the pupils responded neither to light nor convergence. And the normal condition of both pupils was found by Berger in only 4 among 109 cases (Knies). The latter writer has also called attention to the fact that in myosis of spinal origin mydriatics are less effective, while in spinal mydriasis the action of myopics is diminished. In examining for the presence of the light reflex, Swanzy calls attention to the importance of avoiding such cutaneous irritation as is apt to result from touching the skin of the face or lids, and recommends that the test be made in a darkened room.

Paralysis of accommodation is rare. When found, it is generally in association with mydriasis and in the late stages of the disease.

What is sometimes termed sympathetic ptosis, but is really a slight drooping of the upper lids not due to paralysis of the third nerve, is occasionally observed in association with myosis.

Another somewhat rare sympathetic symptom, but which Berger claims to have seen in half of his cases, is epiphora. It is attributed to disturbed lacrymal secretion and imperfect action of the orbicularis palpebrarum. Berger also calls attention to imperfect closure of the eyelids, with fibrillary twitchings of the orbicularis muscle, and to reduced intra-ocular tension, which he attributes to paralysis of the sympathetic. Other writers seldom mention these symptoms.

TROPHONEUROSES.

Acromegaly sometimes causes great thickening of the borders of the orbits, and this may be increased by dilatation of the frontal sinuses. The lids may become hypertrophied and brown in color. The conjunctiva, fat, and orbital muscles are at times hypertrophied, and the exophthalmos which occasionally develops may be accompanied by atrophy of the optic nerves.

Hypertrophy of the pituitary body is by some writers considered as a cause, but Marie looks upon it as one of the results of acromegaly. However this may be, the resulting pressure upon the chiasm and

optic tract may lead to optic neuritis or choked disk, or to bitemporal hemianopsia, or narrowing of the visual fields, defective vision, and sometimes even to complete blindness.

Hemifacial hypertrophy is a rare affection in which the eye necessarily bears an important part. The orbit, lids, and eyeball are often greatly enlarged, leading, when the ball is hypertrophied, to exposure of the cornea from difficulty in closing the lids. Knies and Ziehl have each reported a case in which the eyeball took part, producing a high degree of myopia and, in the case of the former observer, extensive choroidal changes.

Progressive facial hemiatrophy, which begins with unilateral irritation of the cervical sympathetic and later passes into paralysis, involving also a lesion of the trigeminus, produces ptosis, myosis, and enophthalmos, with oculopupillary irritation and paralysis. There is progressive thinning of the skin of the eyelids, and the brows and lashes turn gray and fall out. Retraction of the lids often leads to exposure and disease of the cornea. Kalt observed choroiditis and myopia, and Ruhemann reported ptosis, divergent strabismus, cataract, impaired mobility of the eye, and contracted pupil (Knies).

INJURIES TO THE BRAIN AND SPINAL CORD.

Injuries to the brain produce a variety of eye symptoms which may be classified as (*a*) those which are the direct effect of traumatism of the cortex, nuclei, tracts, chiasm, or optic nerves, and (*b*) those which result from hemorrhage, meningitis, and abscesses of traumatic origin. Under their respective heads the eye symptoms which follow the conditions mentioned in the latter group (*c*) have already received consideration.

Well-defined and clear-cut localizing symptoms may occasionally be observed in gunshot and punctured wounds. A wound of the cortex, if in the occipital region, may be followed by cortical blindness or hemianopsia with conjugate deviation and nystagmus; but fractures, especially fractures of the base, are apt soon to be complicated by hemorrhage and meningitis which obscure the symptoms. Not infrequently, however, in fracture at the base which does not necessarily require a blow of great force, if in the proper direction, one or both of the optic foramina are involved, lacerating the optic nerve, and we have immediate blindness with loss of pupillary reaction, even before inflammatory symptoms have developed. This is, of course, soon followed by atrophy. Both Knapp and Knies have reported such cases, and the latter author records one reported by Taffier in which fracture of the frontal bone was followed by nasal hemianopsia evidently, so he states, due to involvement of the anterior angle of the chiasm. Such traumatisms may also involve the motor nerves, especially the facial, producing lagophthalmos and the abducens, causing paralytic convergence.

Compression of the brain and concussion both cause pupillary changes. In the former we have mydriasis without reaction to light and, in rare instances, congestion and œdema of the papilla, while in the latter Hutchinson describes a degree of sluggishness in the pupillary movements, and Knies refers to the not infrequent occurrence of nystagmus, which he attributes to "cortical inactivity or interference with the conduction of the innervation impulse which starts from the cortex." Concussion of the brain is followed by no ophthalmoscopic change, although, as Gowers has suggested, simple concussion of the nerve and retina may probably cause loss of sight and slow atrophy. Contusions and lacerations of the brain and hernia cerebri may be accompanied by neuritis.

Injuries of the spinal cord rarely cause organic disease of the eyes. No changes in the fundus were found in 17 rapidly fatal cases observed by Allbutt, although among 13 chronic cases he reports 8 as having exhibited more or less hyperæmia of the papilla. He never found true optic neuritis (Knies). When the injury is in the lower cervical or upper dorsal region, a lesion of the sympathetic may lead to vaso-motor and trophic disturbances and inequality or contraction of the pupils.

While in rare instances optic neuritis and atrophy of the optic nerve have been reported, in considering them we should bear in mind the fact that spinal injury may be complicated with injury of the brain, and thus account for the symptoms observed in the eyes.

Various ocular symptoms are of frequent occurrence in concussion of the spine, but it is not usually easy to establish the existence of a real organic lesion in such cases. This is especially true of railway spine, or what by some writers has been termed concussion of the spine; but even where this is the case it is difficult to find records of well-authenticated instances in which it has led to organic disease of the eyes. The neurotic symptoms are often very marked, and, as we may even sometimes find hysterical contraction of the visual fields, it is important, as Swanzy has pointed out, that in our ophthalmoscopic examinations we carefully avoid interpreting what may be a physiological variation in the color of the disk as indicating an organic lesion of the optic nerve.

DISEASES OF THE NERVES.

Multiple neuritis, which is a not infrequent effect of poisonous and infectious diseases, may affect the optic nerve, producing axial neuritis, central scotoma, disturbances of color perception, etc., such as are described in the section devoted to toxic amblyopia. It may also affect the motor nerves, producing partial or complete paralysis.

Knies calls attention to the fact that in amyotrophic paralysis of the arm, when the nerve roots or spinal cord are involved, if the oculopupillary fibres from the cord to the sympathetic become im-

permeable we may have ptosis, myosis, and enophthalmos on the same side.

Inflammation of the first branch of the trigeminus may lead to herpes, which is considered elsewhere.

In **trigeminal neuralgia** the ocular branches are sometimes scarcely involved, but they often do take part, leading to injection, lacrymation, photophobia, and pain in the eye. Inflammation of the ocular branches if severe is quite constantly reflected to the other branches of the trigeminus.

Paralysis of the trigeminus is apt to lead to neuroparalytic keratitis if an abrasion of the cornea occurs and the element of infection is added.

A large proportion of the nervous phenomena which appear as reflex expressions of the accommodative and muscular strain, made necessary by errors of refraction and muscle imbalance, manifest themselves in the superior branch of the trigeminus. And in every case of obstinate trigeminal neuralgia a careful attempt should be made to discover and correct all such errors.

Paralysis of the ocular branch of the facial nerve is followed by lagophthalmos, which may be present in any degree, and, if marked, is apt to be the cause of secondary conjunctivitis and keratitis as the result of exposure.

Tonic and clonic spasm of the orbicularis muscle may be cortical or nuclear in origin, or a reflex symptom of peripheral irritation.

CHAPTER XIV.

GENERAL PREPARATION FOR OPERATIONS UPON THE EYE.

By CLARENCE A. VEASEY, A.M., M.D.

The general principles of aseptic and antiseptic surgery should be employed in operations on the eye, as well as in operations upon other portions of the body, with the exception that the strong germicidal solutions which may be used elsewhere are here, as a rule, not permissible.

PREPARATION OF THE PATIENT.

If time permits, it is important that the patient's general health be placed in the best possible condition prior to the performance of any of the major operations. Naturally, this does not apply in those emergency cases demanding immediate attention; but in other cases the surgeon will be well rewarded for attending to any of the details that will improve the physical or mental condition of the patient. Should diabetes or albuminuria be present, they are grave complications, to be sure, but by no means contraindicate operative procedure, as was formerly thought to be the case. If, however, a course of medicinal and dietetic treatment will improve materially the nephritic condition, the chances for ultimate success in any operation upon the eye will thereby be markedly increased. If any acute pulmonary disease is present, it is wiser to postpone operation until after its subsidence; and again, if there is any chronic pulmonary affection it is not only necessary to improve the condition as much as possible, especially if there is any exacerbation, but it is also of the utmost importance to allay any cough that might give rise to complications during or following the operative procedure. For the latter purpose, in addition to the older remedies, heroin has been much employed of late, and in doses of one-sixteenth to one-twelfth of a grain, frequently repeated, has proved of value, as has also the following spray:

Menthol crystals,		
Pulv. camphoræ.	āā	gr. xxx.
Liq. petrolati,		℥j.

M. To be used locally in atomizer or nebulizer.

The condition of the heart and bloodvessels should also be carefully investigated. If arterio-sclerosis is present, such drugs as will reduce arterial tension, and thereby lessen the probability of hemorrhage, intra-ocular or extra-ocular, should be administered.

Appropriate remedies and exercises should be employed for valvular cardiac affections; and if the patient be a "bleeder" and an operation be determined upon, all possible means for lessening the liability to excessive hemorrhage should be adopted. For this purpose the administration of the calcium salts and of gelatin has proved of value. The latter is not only employed hypodermically, but the eating of large quantities is highly recommended in the condition of hæmophilia.[1]

It is especially important that there be no suppurative disease of the conjunctiva or lacrymal sac in those cases requiring the opening of the eyeball. These conditions should be treated as long as it is necessary to rid the patient of them, both by topical applications and intranasal medication, and, in persistent dacryocystitis resisting prolonged treatment, it is sometimes even advisable to close the lacrymal punctum by means of the actual cautery.

On the day preceding the operation the patient should take a warm bath, including a shampoo for the head and beard, guarding against any exposure to draughts of air. A teaspoonful of compound licorice powder, or a dose of some other laxative, to be followed the next morning by a saline or enema, will place the patient's bowels in the best condition for rest and quiet after the operation.

Fig. 343.

Bottle and irrigator for boric acid solution.

Preparation of the Region of Operation. An hour or two preceding the operation the skin of the eyelids and surrounding parts is thoroughly cleansed with soap and water, followed by alcohol, and then by a solution of mercuric chloride (1: 5000). Particular attention should be given to the eyebrows and ciliary margins, and at the same time care should be exercised not to irritate the conjunctiva by permitting the soap or alcohol to come in contact with it. The conjunctival cul-de-sac is next freely irrigated, either with a sterile boric acid solution (gr. x to ʒj), sterile saline solution (normal strength), or with a solution of mercuric chloride (1: 5000). The eyelashes should be rubbed quite hard, as it is here that many micro-organisms lurk, after which the eye and surrounding parts are covered with a sterile dressing until the surgeon is ready to proceed with the operation. It is also advisable, as advocated by Lippincott, to spray the nares three or four times a day for a couple of days preceding any

[1] For a résumé of the literature and directions as to the use of gelatin, consult a paper by Dr. Joseph Sailer, Therapeutic Gazette, August, 1901.

operation upon the eyeball with a solution of permanganate of potassium (1: 2000), as ample experience has shown that the procedure materially lessens the liability to infection of the wound or to inflammatory processes after operation.

Bacteriological investigations have shown that pathogenic microorganisms are present in every conjunctival cul-de-sac and on the lid margins, and that germicidal solutions sufficiently strong to destroy them also produce injury to the delicate epithelial layer of the cornea, sometimes resulting in permanent loss of a portion of the visual acuity, as well as giving rise to considerable irritation of the conjunctiva itself. We are, therefore, obliged to content ourselves with the removal of as many bacteria as possible, and the diminution of the vitality of those remaining, by forcibly flushing the conjunctiva and scrubbing the lid margins with some of the solutions described.

Immediately preceding the operation the conjunctiva is again flushed with the boric acid or mercuric chloride solution, the lid everted, and the conjunctival surface wiped with a pledget of absorbent cotton moistened with the same solution. It is also well to wipe very gently that portion of the surface of the cornea in which the puncture is to be made in those operations in which the eyeball is to be entered. In all office operations in which the surgeon is ready to proceed at once the temporary dressing is dispensed with.

PREPARATION OF THE SURGEON AND ASSISTANTS.

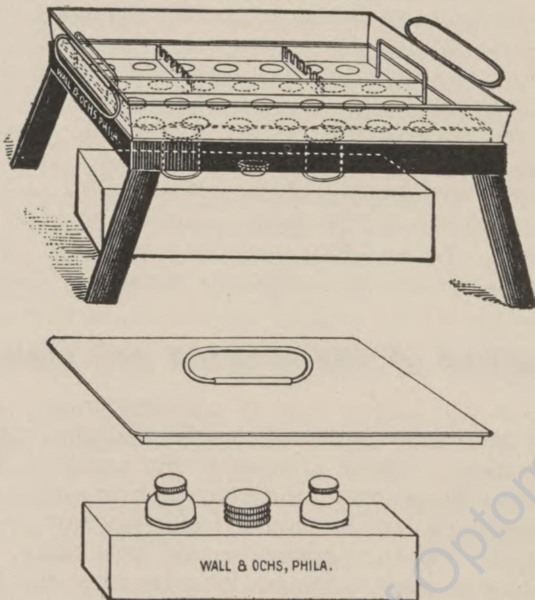
The hands of the surgeon and all assisting him in the operation are scrubbed with soap and warm water, the finger-nails receiving particular attention. After immersing the hands in alcohol for a moment they are dipped in a solution of mercuric chloride (1: 1000), after which nothing should be touched that has not previously been rendered aseptic. If the assistant is one with whom the surgeon is unaccustomed to work, he should be advised of the details of the operation and the order in which the instruments will probably be required. In operations upon the bulb, the assistant should be instructed as to the method of removing pressure from the eyeball by lifting the speculum, and also as to the probable procedures in case of complications. The nurse should be prepared to render any assistance required, and neither surgeon nor assistants should have handled septic cases for some hours before operating. If several cases have to be operated upon at the same time, and some of them are septic, these should be taken last.

PREPARATION OF THE INSTRUMENTS, SPONGES, SUTURES, LIGATURES, AND DRESSINGS.

Instruments. All the instruments to be employed, with the exception of the cutting instruments with very fine points (cataract knives

and needles, keratomes, etc.), should be scrubbed thoroughly with soap and warm water, particular attention being given to the joints and rough parts. It is better to do this immediately after, as well as before each operation. They are then placed in a steam sterilizer for ten minutes, or else boiled from three to five minutes in a 1 per cent. solution of carbonate of sodium, the latter preventing them, to some extent, from rusting, and preserving their cutting edges. From the sterilizer the instruments are immersed in absolute alcohol, where they remain until the surgeon is ready to proceed, when they are transferred to sterile water or sterile saline solution, to cleanse

FIG. 344.



Veasey's portable sterilizer.

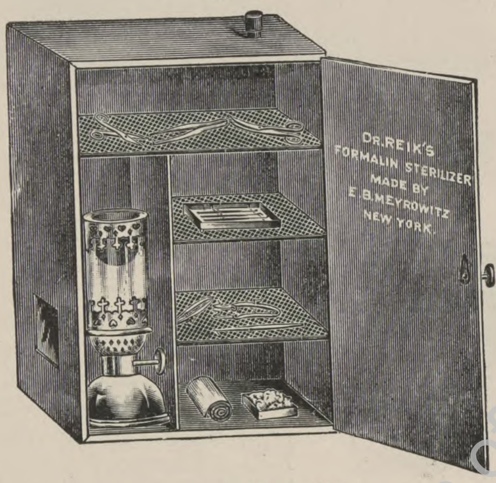
them from the alcohol, which is irritating to the eye. In the operating-rooms of hospitals a steam sterilizer is usually found, but in private practice it is necessary, as a rule, to employ the boiling method. For this purpose the writer's portable sterilizer (Fig. 344) is very convenient. After the instruments have been boiled the perforated tray containing them can be transferred to the alcohol, and then to the water or saline solution, no instrument being touched until the operation begins.

Experience has shown that the delicate cutting instruments cannot be treated in the same manner as the coarser instruments without destroying to a large extent their cutting qualities. They are, therefore, first wiped with sterile cotton moistened with absolute alcohol. The cutting edges and points having been carefully inspected for rust or blood clots, are then wrapped with sterile cotton and held

for a moment in boiling water, after which they may be placed in absolute alcohol or wrapped in sterile absorbent cotton until required for use. Immediately before using them they should again be dipped in boiling water for an instant.

Many different methods to obtain the same results are employed by different ophthalmic surgeons. For example, some do not employ absolute alcohol, but place the instruments after boiling in a 3 per cent. solution of carbolic acid, or a 1: 1000 solution of formaldehyde. Others transfer them at once from the sterilizer to sterile water. Still others sterilize by means of formaldehyde gas, employing specially devised sterilizers for the purpose. The one here illustrated (Fig. 345) was devised by Reik, of Baltimore, and is a very excellent one for this method. Before beginning an operation, it is

FIG. 345.



Reik's formalin sterilizer.

always a good procedure to arrange the instruments in the order in which they will probably be required. This can be done when placing them in the sterilizing tray, and any one can then be picked out more quickly should complications arise during this operation.

Sponges. In ophthalmic operations ordinary sponges may be used prepared by the usual methods in vogue among general surgeons, but small pieces of sterile gauze, or small pledgets of sterile cotton, are preferable. In deep-seated operations in the orbit it is more satisfactory to have the gauze wrapped on the ends of sterile sticks or probes, so that, in sponging, the parts may be more readily reached and the assistant's hand will not obscure the field of operation.

Sutures and Ligatures. Catgut, either plain or chromicized, and fine silk are employed as sutures and ligatures in ophthalmic operations, and the methods of their preparation do not differ from those in use in general surgery. The silk may be either white or black,

but the latter is to be preferred if it is to remain in position for some time, as it is more readily located for removal. After the sutures are removed from the solutions in which they usually are kept they are placed in absolute alcohol until required, when they are rinsed in sterile water. The same procedure applies to ligatures.

Dressings. The dressings to be placed upon an eye after an operation necessarily differ according to the nature of the operation and the exigencies of the case. In extensive operations about the lids they do not differ from those employed in surgery elsewhere—viz.: a protective, pads of sterile gauze, absorbent cotton, and a roller bandage. The gauze may have been made sterile by heat, or soaked in a solution of mercuric chloride (1: 1000 or 1: 2000) or other germicide, and dried. The bandage should also be sterile, and gauze is to be preferred in preparing it, as it retains its position much better than if made of flannel. The dressing may be either dry or wet. In the latter instance it is soaked in some solution before placing in position, usually mercuric chloride (1: 5000), boric acid (gr. x to ℥j), or sterile normal saline solution, and the excess squeezed out with the hand.

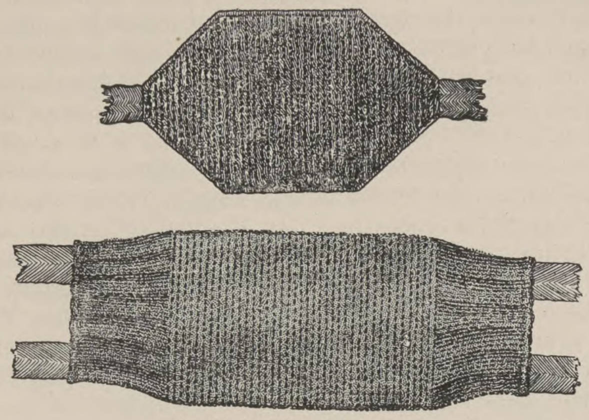
Fig. 346.

Ring's eye mask.

In the majority of ophthalmic operations the following method of dressing the eye will prove satisfactory: A few layers of sterile gauze two and one-half inches in diameter are moistened with a solution of mercuric chloride (1: 5000), and placed over the closed eyelids. Over this is placed sufficient sterile absorbent cotton to fill in the depression made by the orbital ridge and the nose. These are fastened securely in position by a few strips of isinglass plaster. If the patient is quiet, this is sufficient; if he is restless, however, or untrustworthy as to passing his fingers beneath the dressings, or lifting it in an attempt to see, it is safer to add the figure-of-eight bandage. Indeed, in many cases, as after cataract extractions or iridectomies, it is an excellent plan to place over the above dressing a protective mask such as that shown in Fig. 346, which was devised by the late Dr. Frank W. Ring, of New York, and is made of papier-maché. Other masks made of wire or aluminum are employed by some surgeons. In place of the figure-of-eight bandage in some cases, and especially in those who are continuing their vocations, a modified *Liebreich bandage*, which is knit of black zephyr and fastened in position by a tape at each end, will be very acceptable. It is made either single or double, according to whether it is required for one or for both eyes. A *pressure bandage* is not applied differently from that described, except that sufficient cotton is so placed over

the gauze pads that, when the bandage is applied, firm pressure upon the eyeball is made. A *ring dressing*, such as is employed some-

FIG. 347.



Modified Liebreich bandage.

times after skin-grafting, is made by surrounding the whole field of operation with a ring of sterile gauze sufficiently thick to prevent the dressings from coming in contact with it. *Eye shades* are sometimes employed after the dressings have been discarded, and should be firm, light, and so shaped that sufficient air can enter behind them to keep the eye cool and dry. (Fig. 348.) A Buller shield may be employed in certain cases as a protective dressing. Occasionally, as in tuberculous disease of the orbital bones, it is necessary to employ iodoform dressings about the eye, but these do not differ in any respect from similar dressings employed elsewhere.

FIG. 348.

Eye shade.

ANÆSTHESIA.

General Anæsthesia. In the great majority of ophthalmic operations local anæsthesia is sufficient; but in a few, such as enucleation of the eyeball, operations upon severely inflamed eyes, removal of orbital growths, plastic operations upon the lids, and in most of the operations upon children or very nervous individuals, general anæsthesia is required. Ether is preferable to chloroform, except, perhaps, in patients affected with chronic bronchial diseases, in whom it is liable to give rise to an attack of pneumonia. Bromide of ethyl is sometimes employed, and at the present time is attracting considerable attention among French surgeons, but by most operators it is considered to possess no advantage over ether or chloroform.

Nitrous oxide gas may be employed advantageously in short operations in very nervous patients, as in the dilatation of strictures of the lacrymal duct or in the removal of a chalazion.

Local Anæsthesia. For the purpose of local anæsthesia in operations upon the eye, the hydrochlorate of cocaine is employed probably more generally than any other drug, although within recent years a number of new local anæsthetics have been introduced, each of which has its advocates. The hydrochlorate of cocaine is employed ordinarily in a 2 to 4 per cent. solution, and even as strong as 10 per cent. by some surgeons, and, as it produces some softening of the corneal epithelium, the eyelids should remain closed after its instillation. For superficial operations, one instillation usually suffices, the operation being performed five minutes later; but in deeper operations, for example, iridectomy, extraction of the cataractous lens, and tenotomy, three instillations should be employed at five-minute intervals, and the operation begun fifteen minutes after the first instillation has been made. By following this method, the cocaine has sufficient time to reach the deeper structures of the eye, and much less pain will follow the seizure of the iris or tendon than otherwise. Operations should be completed either within twenty minutes from the time of the last instillation, or other instillations made to prolong the effect. For the removal of a Meibomian cyst, the hypodermic injection of a drop or two of a 2 per cent. solution in the immediate neighborhood of the cyst will render the operation far less painful than will several instillations.

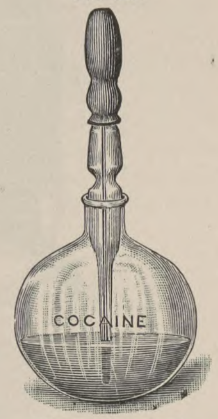
Hydrochlorate of eucainc "A" and hydrochlorate of eucaine "B" are sometimes used as local anæsthetics, although the first is employed by no means so frequently as formerly, as the latter is less irritating and less toxic. The advantages claimed for eucaine "B" are that it is one-fourth as toxic as cocaine, does not affect the heart, does not produce mydriasis, nor affect the accommodation. The disadvantages are much more congestion and bleeding during and after operation, and frequent sloughing of the tissues when employed hypodermically. For producing local anæsthesia about the eye, 2 per cent. solutions are recommended.

Tropacocaine in 3 per cent. solution is employed by some surgeons (Schweigger, Silex), who claim more rapid anæsthesia than with other drugs of this class. The effect also wears off more quickly.

Holocaine is one of the newest members of this group, and is used in from 1 to 2 per cent. solution. It is claimed that it does not affect the corneal epithelium, produce mydriasis, nor impair the accommodation, and that it produces anæsthesia more quickly than cocaine; the anæsthesia, however, does not last so long. It is preferred to cocaine by some surgeons (Knapp, Derby) in all operations in which local anæsthesia is used. As it possesses some bactericidal action and does not affect the corneal epithelium, it should be given the preference in corneal affections in which local anæsthesia is required, and in the removal of foreign bodies.

Inasmuch as most of these solutions, as well as other alkaloidal solutions employed in the eye, present excellent media for the growth of various fungi, they should be sterilized thoroughly before being used in operative procedures. This may be done by boiling or by preparing the solutions in some antiseptic medium; for example, 1:5000 mercuric chloride, or 1:1000 tricresol (E. A. de Schweinitz). The use of a saturated solution of boric acid will not prevent the growth of fungi, but will prolong somewhat the period prior to their appearance. For sterilization by boiling, the Stroschein flask is very convenient. (Fig. 349.)

FIG. 349.



Stroschein's flask.

Infiltration Anæsthesia. This method of producing local anæsthesia, as suggested by Schleich, consists of the *intracutaneous* injection of the following solution:

Cocainæ hydrochlorat.,	gr. j.
Sodi chloridii,	gr. j.
Aquæ destillata,	℥j.—M.

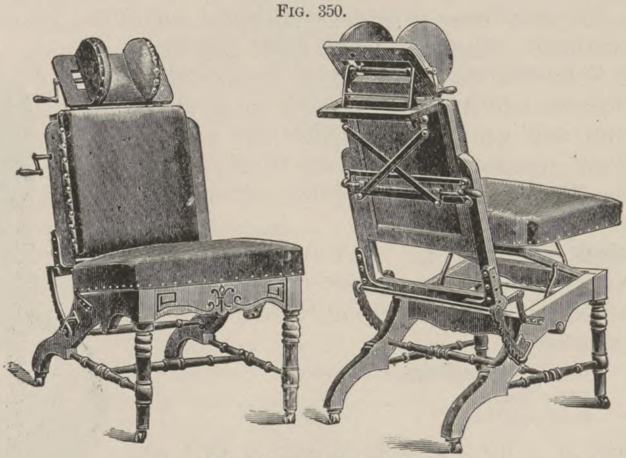
By means of a hypodermic syringe a drop or two of the solution is injected into the skin, resulting in the production of a small wheal. Another injection is then made at the edge of the first wheal, and the procedure repeated until the area desired has become anæsthetized. The anæsthetic area, however, is limited strictly to the wheals thus produced, and, on account of the vascularity and looseness of the tissues of the lids, the method is of infrequent use in ophthalmic practice.

POSITION OF PATIENT AND OPERATOR.

The position of the patient during operation is perhaps more a matter of convenience to the surgeon than of importance to the patient, except in rare instances. In major operations most operators prefer to have the patient in a reclining posture, preferably in the same bed in which he is to remain during convalescence; others employ an operating-chair, the patient being in a half-sitting and half-reclining position during the operation. In most operations it is probably a matter of little consequence which plan is adopted, but in iridectomy and in extraction of the cataractous lens it has always seemed to the writer more convenient and less risky to operate upon the patient in his own bed if suitable illumination could be obtained. Ordinarily the patient should be reclining upon his back, his head resting upon one or two hard pillows, with the face directed upward, his eyes being slightly lower than the level of the forearm of the surgeon when it is extended a little below the horizontal, as this is the position least tiresome to the operator. In modern hospitals the beds can be moved on rubber-tired trucks wherever desired, and the jar of transferring the patient from the

table or chair to the bed is thereby avoided. Should a chair be employed, the one recommended by Knapp is very satisfactory (Fig. 350); but for minor operations in the office an ordinary arm-chair with

FIG. 350.



Knapp's operating-chair.

FIG. 351.

Operating mask for practising on animals' eyes.

a low back, over which is placed a small pillow for the patient's neck and head to rest upon, will be found to answer the purpose fairly well.

The position of the operator may be either behind or beside and somewhat in front of the patient. If he is ambidextrous, he may stand behind in all operations; if not, he will be obliged to stand behind in certain operations upon the right eye, and beside the patient in the same operation upon the left eye; for example, in making corneal sections in the extraction of cataract. If he will accustom himself to stand behind the patient in as many operations as possible, however, it will be found much more convenient, as he may operate upon either eye without shifting his position.

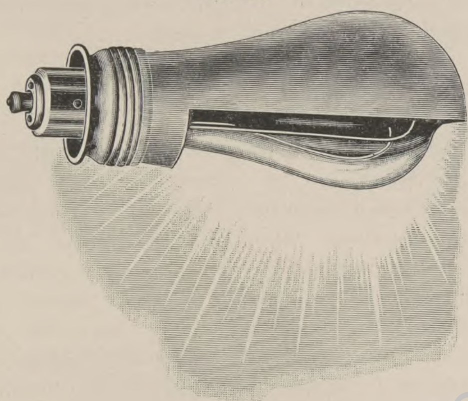
Knapp has truly said that ambidexterity is not a gift of nature, but must be acquired. This can be done only by frequent practice. To most of us it is quite natural to manipulate the instruments with one hand only; the other must, therefore, be educated. This can best be done by practice upon the eyes of animals in operating masks (Fig. 351), those of the pig being the best, as sufficient quantity can always be obtained. The methods of practice have been given elsewhere.[1]

[1] See the writer's book, Ophthalmic Operations as Practised on Animals' Eyes.

ILLUMINATION.

In most of the operations upon the eye it is imperative that the illumination be of the best. It matters not whether it is ordinary daylight or artificial light from an Argand burner or electric bulb. It should come from the side of the eye to be operated upon, and should be the brightest possible, barring direct sunlight. That obtained from a small space is the most satisfactory, as annoying reflections are thereby avoided. If artificial light from an Argand burner is employed, the assistant throws it upon the eye by means of a condensing lens, thus increasing its brilliancy; if from an electric bulb, the light should be covered with a reflector. The latter is a very convenient method of illumination in cases in which the arti-

FIG. 352.



Electric bulb with reflector.

ficial light is employed, as it may be attached to any outlet from the street current, and with sufficient wire can be carried to the most remote parts of the operating-room. (Fig. 352.)

It is also of great importance that the vision of the operator be good. The eye is a very small organ, and many of the operations require delicate manipulation in small spaces, so that good vision and illumination are indispensable. Operators having refractive errors requiring correction for distinct near vision should wear that correction while operating. Various magnifying glasses have been suggested from time to time by different surgeons—Jackson, Berger, and others (Fig. 4, Chapter I.)—to improve the vision in operative work, even in those who have no refractive error, but these have not seemed to the writer to possess any advantage over the ordinary correcting lenses in those whose media are perfectly clear.

TIME OF PERFORMANCE.

Operations may be performed at any hour of the day or night, or at any season of the year. With proper illumination, just described, cases first seen at night or on very cloudy days, and requiring immediate operative interference, need not be postponed. The season of the year influences results in those operations requiring more or less prolonged confinement in bed only in so far as it may increase the debilitated condition of the patient. Obviously, therefore, if avoidable, very stout persons should not be operated upon in excessively hot weather, nor those affected with nephritic or pulmonary diseases in extremely cold weather. In most cases it is probably better to operate, if possible, early in the morning, while fresh and before other cases have been handled. In the operation for cataract, however, it is thought by some surgeons to be better to operate in the afternoon, as the few hours of smarting which usually follow this operation will be succeeded by a night's sleep and rest, so necessary for the early union of the corneal wound.

AFTER-TREATMENT.

It is manifestly impossible to give any hard-and-fast rules concerning the after-treatment of operative cases applicable to all alike. The special requirements of the more important operations have been given elsewhere when the technique of the operative procedures has been described. In general, however, if there is much pain following an operation or the patient is restless, an anodyne should be administered. Sleep and rest can usually be obtained, if pain is not present, by the administration of 15 grains of trional. If pain is a prominent symptom, a hypodermic injection of morphine may be given. The patient should not be permitted to lie in one position any longer than absolutely necessary, especially upon the back, for fear of hypostatic congestion of the lungs. If there is any history of hemorrhage having followed a former operation, the head of the patient should be kept high by means of several pillows in addition to elevation of the head of the bed. Patients accustomed to the habitual use of considerable quantities of spirituous liquors should not have these entirely withdrawn, but should be served in moderation. In operations requiring the eyeball to be opened or in extensive operative procedures, it is better to keep the patient on soft diet for the succeeding twenty-four to forty-eight hours. As a rule, the dressings should be changed daily, and the eyes carefully inspected and cleansed with warm boric acid lotion until they are no longer required; and eyes that have been bandaged for some time should gradually be accustomed to the light. If the bowels do not move of themselves in three or four days, a laxative should be administered, and in all operations upon the bulb straining at stool should carefully be guarded against.

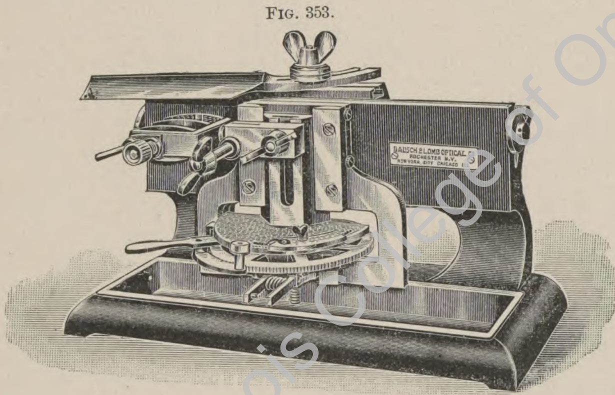
CHAPTER XV.

THE TECHNIQUE OF THE PATHOLOGICAL AND BACTERIOLOGICAL EXAMINATIONS OF THE EYE.

By EDWARD A. SHUMWAY, B.S., M.D.

The study of the pathology of the eye, in certain sections, has not kept pace with that of general pathology, a fact that is due perhaps to the almost entire silence upon this subject of works on general pathology and pathological technique. In no part of the body, however, are so many different tissues intimately related in a small space, and the additional interest which the microscopic study of the varied pathological conditions brings to our clinical observations, well repays the efforts made to master the technique that is required. An elaborate outfit is not necessary. A good microscope, with an oil-immersion lens, if bacteriological examinations are to be made, and a microtome, for holding the knife in making sections of the embedded

Fig. 353.

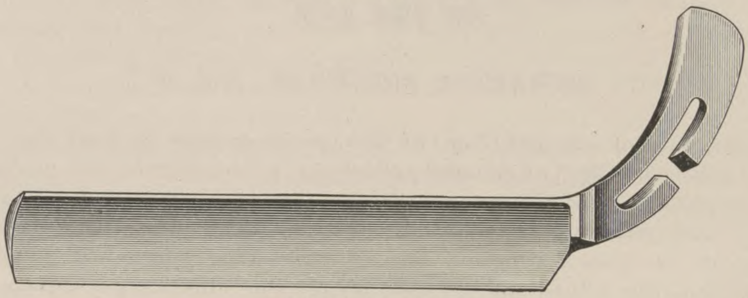


Medium laboratory microtome.

tissue, are the first essentials. An expensive microtome may be dispensed with. The Schanze model (Fig. 353), made by the Bausch & Lomb Optical Company, is an excellent instrument. Good section-cutting depends more upon the success in embedding and upon the condition of the knife than upon costly microtomes. The best knives are made by Walb, in Heidelberg, and a fairly heavy one, with a

cutting edge from 16 to 20 cm. long, should be selected. (Fig. 354.) Aside from these larger instruments, we need a pair of fine forceps (preferably curved), needle-holders, spatulas (one broad and one narrow), staining dishes, cover-glasses, slides, etc. For many sections the ordinary slide, 1 x 3 inches, is sufficiently large, but for sections of the entire eyeball, slides $1\frac{1}{2}$ x 3 inches, or the German size, 70 x 35 mm. ($1\frac{3}{8}$ x $2\frac{3}{4}$ inches), may be ordered. Convenient sizes for cover-glasses are 18 mm. square; 21 x 26 mm. for sections of

FIG. 354.



Knife for microtome.

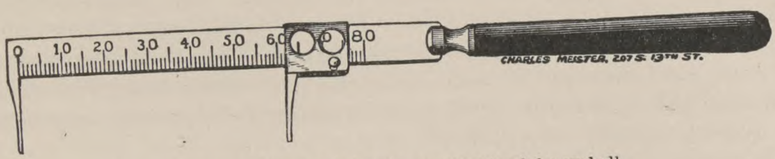
one-half of the eyeball, and 28 mm. square for the entire ball. Larger ones, 28 x 32 mm., are occasionally useful, and when still larger forms are necessary isinglass may be obtained in sheets and cut to the proper size.

Obtaining Material. Normal eyes are difficult to obtain, but may be secured occasionally in resections of the upper jaw, and very satisfactory specimens are furnished when the eye is enucleated for small malignant growths of the anterior part of the eyeball or of the optic nerve. Many interesting conditions require enucleation, and pathological alterations of the conjunctiva may be studied by removing small bits of the tissue after cocaine anæsthesia. In post-mortem examinations the removal of the eyes is rarely permitted; but if the skull has been opened, the posterior halves of the eyes may be secured by breaking through the roof of the orbit, dissecting away the fat, and carefully cutting through the sclera in the equatorial direction with a sharp pair of scissors.

After the material has been obtained, it should be placed in a fixing fluid as soon as possible, in order that the fine changes, which take place in the nervous structures of the eye, may be prevented, and the tissues preserved in approximately the same condition as in life. The exact time for the earliest appearance of post-mortem changes in the retinal ganglion cells has not been determined for the human eye. Birch-Hirschfeld, however, found decided changes in rabbits' eyes two hours after death, and it is fair to assume that if a much longer time has elapsed, the human eyeball can be of little value for a study of the ganglion cells, although it may still be useful in determining changes in other less susceptible portions.

Preparation of the Eyeball. On removal of the eye the measurements of its diameters should be made, and any peculiarities in its external appearance carefully noted. The measurements may conveniently be obtained by means of an instrument like the one shown in Fig. 355, which was designed for measuring interpupillary distances. They should include the antero-posterior diameter and the vertical and horizontal diameters at the equator. The side of the head from which the eye was removed should also be noted. The optic nerve enters the eyeball on the nasal side of the posterior pole, so that if the long axis of the cornea is held in a horizontal

FIG. 355.



Instrument for obtaining measurements of the eyeball.

direction, and the optic nerve directed toward the imaginary fellow eye, the problem of orientation later should be a simple one. If this fact is not recorded, we must rely on the appearance and insertion of the oblique muscles, provided that they are still present. The inferior oblique tendon is more fleshy, and is attached farther away from the cornea than the superior. Both are inserted on the temporal side of the corresponding rectus muscle. Hence, if the long axis of the cornea is held again in the horizontal plane, with the superior rectus up, the attachments of the obliques will indicate the temporal side of the eyeball, and the side from which it was removed. The position of any ulceration or opacity of the cornea should be sketched, and the presence of a coloboma or other peculiarity of the iris noted. If an intra-ocular tumor is suspected, it may be located by noticing a difference in resistance to pressure, or by looking through the pupil while the eye is held before a bright light. Ordinarily, sections are made of the antero-posterior diameter of the eyeball, so that the eye should be divided in this direction. This, however, should be done after the fixation and hardening, in order to avoid distortion of the halves.

Fixation and Hardening. Unless some special examination is required (see method for the retinal ganglion cells, page 678), we may confine ourselves to the use of two solutions—formalin and Müller's fluid.

Müller's fluid consists of: potassium dichromate, 2.5 gm.; sodium sulphate, 1 gm.; distilled water, 100 gm. The solution should be used in considerable quantity, and the bottom of the jar covered with cotton, so that the fluid may have ready access to all portions of the eyeball and the eye not injured in transportation. It

is unnecessary to make an opening in the eyeball, as the fluid penetrates rapidly. The specimen should be kept in the dark to prevent the formation of precipitates, and the fluid should be changed daily until it no longer becomes cloudy. At the end of six weeks, at ordinary room temperature, or after two weeks in the incubator (37°–40° C.) the eye should be washed thoroughly in running water for twenty-four hours, and then hardened in gradually ascending strengths of alcohol. Müller's fluid is of special value when it is desirable to employ the Weigert stain for the nerve sheaths, and even when other methods for fixation are used, it is well, if the length of the nerve permits, to cut off a piece and place it in Müller's fluid for cross-sections. It is also the best medium for the examination of the lens, as formalin causes this structure to shrink. Its disadvantages, however, are that it disturbs the chromatin elements of the cells, and is not, therefore, suitable for the study of nuclear structures; it, moreover, does not at once check the post-mortem growth of organisms, and makes the subsequent staining for bacteria, especially for tubercle bacilli, very difficult.

Formalin is a 40 per cent. solution of formaldehyde gas, and should be diluted with 9 parts of water for ordinary use. Stronger solutions are apt to cause black precipitates in the sections, particularly in the presence of blood. The eyeball should not remain in the solution longer than forty-eight hours. The penetration and fixation are very rapid, and the sclera and lens soon become so hard that they are difficult to cut. Subsequent washing with water is desirable, but not absolutely necessary, and the globe is then hardened with alcohol. This should be done slowly, in order to avoid shrinking of the tissues and detachment of the retina as far as possible. Beginning with a 33 per cent. solution, the eye remains for a day each in 33, 40, 50, 60, 70, and 80 per cent. solutions. It is left in 80 per cent. alcohol for several days, and is then ready to divide.

Alcohol should not be used as a fixing agent, as for this purpose it must be of absolute strength, and the rapid withdrawal of water from the tissues, which accompanies the fixation, produces so much shrinking that the eye is very much distorted. It is of considerable value if the sections are to be examined for tubercle bacilli, or in examination of the retinal ganglion cells by the Nissl method, but formalin is almost equally serviceable in these cases. The combination of Müller's fluid and formalin in the proportion of 10 parts of Müller's fluid and 1 part of strong formalin, as suggested by Orth, is a very good one; the swelling of the tissue caused by the one offsets the shrinking produced by the other.

Cutting the Eyeball. If a part of the eyeball is to be preserved as a microscopic specimen, it will usually be cut in a horizontal or vertical plane, which passes through the cornea and optic nerve. If an intra-ocular tumor is present, the eye should be so divided as to show the connection of the growth with the tissue from which it has sprung. Tumors of the anterior segment of the eye can readily be

seen; those of the choroid can usually be located by careful palpation of the sclera. If this precaution is not observed, the section may pass through the cap of the growth as it projects forward, and the very puzzling picture be presented of a round growth lying isolated in the centre of the eyeball. If the entire eye is to be used for microscopic study, the best plan is to make an antero-posterior section well to one side of the optic nerve and the corneal centre. In the subsequent embedding process the retina is apt to detach and sink below the level of the sclera, so that if the eye has been divided exactly through the centre of the nerve and cornea, many of the first sections will not include this membrane, and a portion of the nerve itself may be cut away before the specimen is accurately levelled and good sections are obtained. The macroscopic mounts may, however, be so valuable for demonstration purposes that the division into two halves is unavoidable. If the eyeball has been removed because of an exudative inflammation, and especially if it is atrophic, it may be cut at once without danger of displacement of its contents. For this purpose a sharp brain knife or a good table knife should be used. A razor is objectionable, because its finely honed edge is too easily turned, and because the thick back prevents a smooth section. In a relatively normal eye the lens is so easily displaced that the eyeball should first be frozen. Cutting from the nerve forward, with the cornea pressed against a resisting surface, is less apt to displace the lens than cutting laterally, but it injures the epithelium of the cornea and produces bad artefacts. The freezing necessitates the removal of the alcohol, by immersion of the eye in water for at least twelve hours, until it sinks to the bottom of the vessel. It is then carefully dried, wrapped snugly in oiled silk or rubber tissue, and placed in the centre of a mass of coarsely broken ice and salt, in a box with a perforated bottom, through which the water from the melting ice may be drained. Ordinarily one hour will suffice to freeze the specimen, and it is now cut quickly, on a large cork, with as little sawing motion as possible. The two hemispheres are then allowed to thaw in distilled water. By this method the contents of the globe retain their relative positions and permit of a general description of the conditions present. The half which contains the most important changes should be reserved for microscopic study.

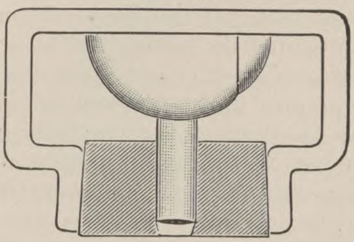
Preservation of the Macroscopic Portion. This may be done in one of three ways: by drying, by mounting in fluid, or by embedding in gelatin.

1. *Dry Method.* The hemisphere is passed again through alcohol in ascending strengths until absolute alcohol is reached. It is then immersed in pure turpentine for several days, and the turpentine is allowed to evaporate slowly. Good, permanent dry specimens are thus obtained. The method, however, is rarely used in this country.

2. *Preservation in Fluid.* For this purpose formalin in 4 per cent. solution is very useful. The eye is placed in the solution immediately after thawing, and may be conveniently mounted in a cup of

the model shown in Fig. 356. It is held in position, face downward, against the flat surface of the cup by means of a glass rod which passes through the rubber cork. The disadvantages of the method are the impossibility of preventing disturbing bubbles of air from leaking in, however much care be taken in the mounting, the danger of subsequent displacement of the contents of the eyeball, which are not firmly fixed in the fluid, and the distortion of the specimen by the pressure of the glass rod, especially if the eyeball is empty. The last difficulty may be avoided by cementing the eye to the base of the cup by means of gelatin. (Gelatin is allowed to swell in water for several hours, the water is poured off, and an equal volume of glycerin is added, and the gelatin melted by heat and filtered. When the eye is to be mounted, the gelatin is melted, applied in a thin coat to the cut surface of the eye, and the latter pressed firmly against the bottom of the cup, which may presently be filled with the formalin.) The advantages of the method are the ease in mounting, the preservation of the natural color of the specimen, and the possibility of utilizing the eye later for microscopic examination, if necessary.

FIG. 356.



Cup for the preservation of macroscopic sections in a solution of formalin.

A modification of Kaiserling's method may also be used with the same eye cups. The eyes are placed in 4 per cent. formalin for twenty-four hours, and are then cut, and the half for the macroscopic mount is wrapped in cotton and placed in solution A, composed of: sodium acetate, 3 gm.; potassium chloride, 0.5 gm.; formalin, 10 gm.; water, 100 gm. It remains in this fluid for four days, and is then placed in 95 per cent. alcohol, which restores the original colors, and after twenty-four hours is changed to solution B, which consists of: potassium acetate, 30 gm.; glycerin, 60 gm.; water, 100 gm. After three or four days the eye is permanently mounted in a fresh solution of the same formula.

3. *Preservation in Glycerin Jelly.* If the eyeball has been hardened in Müller's fluid, it should be washed thoroughly in water for several days, and bleached in a 5 per cent. solution of chloral hydrate until as much of the color is removed as may be possible. It is now placed in a mixture of glycerin and water—at first in the proportion of 1:3, and then of 1:2, for one day each, and is ready for embedding in the glycerin jelly. The latter is made as follows (the strength of the gelatin is greater than that usually recommended): 40 grammes of a fine quality of gelatin (Coignet & Co., Paris, or Conte Fils, Magdeburg, Germany) are covered with 250 c.c of water in an agateware vessel, and allowed to swell. Heat is carefully applied, and the mixture constantly stirred to prevent

burning; otherwise the jelly will have a brownish color. After the gelatin is dissolved, the white of an egg or a small amount of prepared egg albumin (Merck) is added, and the solution is boiled vigorously and filtered through a good filter-paper while hot. The filtrate should have a very pale straw color, and be perfectly clear. To it is added an equal volume of glycerin (C. P.), and 10 c.c. of a 10 per cent. solution of carbolic acid for each 1000 c.c. of the mixture, to prevent the growth of bacteria and moulds. The mounting jar is a plain cup, measuring $1\frac{7}{8}$ inches in diameter and 1 inch deep, with a flat, well-polished base and perpendicular sides. It is filled nearly to the top with the melted jelly, and the eye is immersed with the cut surface up. All bubbles of air are coaxed out of the meshes of the tissue by means of a needle, and those on the surface of the jelly are removed by touching them with a platinum loop which has been heated in a Bunsen burner. The eye must then be turned over carefully, so that no bubbles of air are included, the presence of which may be detected by holding the cup over a hand-mirror. If the eyeball is empty, this requires some skill; but if it is turned with one needle, while a second one pushes in the sclera and holds the cut edge below the surface of the jelly, the difficulty will be slight. The jelly should cover the specimen, but should not fill the cup to the top. The eye is held in a central position by means of a pin which projects through a piece of wood or card-board placed over the top of the cup, and the specimen is placed under a bell-jar or other suitable dish until the jelly hardens. After several days the open top of the cup is sealed by cementing to it a white porcelain disk, and the cup may be mounted in a wooden base. (Fig. 357.)[1] Good glycerin jelly is quite transparent, and fixes the eye firmly, so that there is no danger of subsequent displacement of the eye contents. The one difficulty in temperate climates is the melting of the jelly in very warm weather. This may partly be avoided by turning the specimen cups over in summer, or, better still, by exposing the mounted specimens to the fumes of strong formalin placed in a dish under the bell-jar while the jelly is hardening. Strong formalin added to the gelatin in solution gives it a whitish, semiopaque appearance, which hides the details of the eye but does not have this effect when in a gaseous state. In forty-eight hours the jelly is hardened to a depth of about a quarter of an inch, and this portion will not melt even in a Bunsen burner. If the additional precaution is taken to invert the cups in very warm weather, no difficulty should be experienced in preserving the specimen in good condition.

FIG. 357.

Base for mounting cup.

[1] Wall & Ochs, Philadelphia, have the cups, porcelain disks, and wooden bases in stock.

The Preparation of the Microscopic Specimens. The half for microscopic examination should be searched carefully for the presence of calcareous or osseous deposits, which are not uncommon after a long-standing exudative inflammation of the choroid and in atrophic eyeballs. Such deposits occur most frequently in the surface of the choroid, and will ruin the edge of the microtome knife unless removed. Many solutions are used for decalcification. Müller's fluid has this action, but requires a very long time. Much more serviceable is a 10 per cent. solution of trichloracetic acid, which decalcifies the tissue in a few days, and does not injure its staining qualities. The solution should be changed daily, and the tissue thoroughly washed in water afterward. Nitric acid, in 3 to 9 per cent. solution in water, or in 70 per cent. alcohol, and the following solution, containing phloroglucin, also give good results: phloroglucin, 1 gm.; nitric acid, 5 gm.; alcohol, 70 gm.; water, 30 gm.

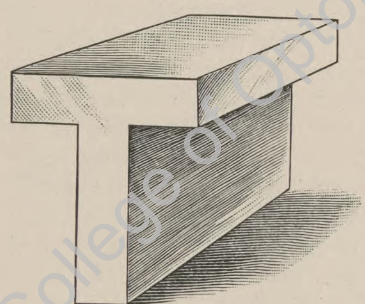
Having freed the specimen from chalk deposits, it is ready for embedding. For this purpose we must have a substance which will infiltrate the tissue thoroughly and will also be sufficiently firm to prevent any motion of the parts as the knife is drawn across. The most satisfactory material for the entire eyeball is celloidin. Photoxylon, which has been recommended as of equal value as celloidin, is still used in Germany, but can no longer be obtained in this country. Paraffin is very useful in cutting small growths, or when very thin sections are necessary, as of the retina, but it does not infiltrate the sclera and lens well. Celloidin is soluble in equal parts of alcohol and ether. It is usually furnished in this country in the form of shavings, in one ounce quantities (Schering), and is dissolved most readily by covering it for twenty-four hours with absolute alcohol, by which it is softened. On the addition of an equal quantity of ether the mass slowly dissolves. It should be of a thick, syrupy consistency for embedding, and a thinner solution, made from the stock solution by the addition of more alcohol and ether, should also be on hand. It should be stored in well-stoppered jars, and may be kept free from possible moisture by placing it, together with the jars containing the absolute alcohol and alcohol-ether, in a large, tightly covered jar, in which there is a quantity of calcium chloride to keep the air dry.

Before the eye is embedded, all traces of water in the tissue should thoroughly be removed. This is done by carrying it again, after thawing, through ascending strengths of alcohol until absolute alcohol is reached. The absolute alcohol is kept best in a large jar, the bottom of which is covered with cupric sulphate which has been thoroughly dried by heat. The copper absorbs water very eagerly, and keeps the alcohol pure ; it should be covered by several layers of filter-paper, or the specimen should be held above it by means of wire gauze netting suspended in the alcohol. From absolute alcohol, after twenty-four to forty-eight hours, the eye is transferred to equal parts of alcohol and ether for twenty-four hours, then to thin

celloidin, and to thick celloidin for at least one day each. The celloidin is now allowed to harden very slowly in a glass dish at least one inch deep, and wide enough to leave considerable space between the eye and the side. All air bubbles are removed carefully, and the dish is covered with a tumbler or similar vessel, one side of which may be raised slightly after twenty-four hours. It is very important that the surface should harden slowly, otherwise large bubbles collect beneath it. The length of time required for the evaporation of the ether varies, but usually it will take three or four days. The celloidin should be loosened occasionally, by a needle, from the wall of the dish, and when it is sufficiently firm at the bottom to prevent the needle from cutting it on slight pressure, the block may be removed by shaking and placed in 80 per cent. alcohol. In this it absorbs water and becomes firm. The best block of celloidin should be only slightly opaque, and its surface should not be easily peeled off by the finger-nail. It may be made still firmer by adding glycerin to the alcohol.

Cutting. After twenty-four hours in alcohol the block is ready for cutting, and may be mounted on a firm object which can be clamped in the microtome. Blocks of pure white pine or maple wood may be used, and if the block is too large to enter the object-holder of the microtome a portion of the underpart may be cut away, as shown in Fig. 358. Vulcanized fibre, used for insulating purposes, may be cut in convenient sizes, and has the advantage of not staining the alcohol. When the block needed is too large, a smaller piece may be fastened to its under surface by means of brass screws or a T-shaped block may be cut as described above. Superfluous celloidin should be trimmed away, and a good flat surface cut parallel to the plane in which the sections are to be made. The base of the celloidin block is dried and then immersed in alcohol-ether for a half-minute until the celloidin is softened. Moderately thick celloidin is poured on the object-holder, and the specimen is mounted in it and held firmly in place for a few minutes, when the entire block is put back into 80 per cent. alcohol to become firm. In cutting, the specimen should be clamped very firmly in the microtome, and should be levelled carefully. The knife should be placed at as acute an angle as possible, and the blade and object kept thoroughly flushed with 80 per cent. alcohol. The knife should be drawn *slowly* and *evenly*, without pressure of the hand downward. The sections are held flat on the blade by means of a light brush held in the left hand, or

FIG. 358.



T-shaped block of wood to hold sections of celloidin with embedded eyeball.

allowed to roll up, and then unrolled when the section is complete. The sections to be stained at once are placed in distilled water; the remainder are put in 80 per cent. alcohol. If serial sections are important, numbered circles cut out of thin paper may be slipped between each section as it is cut, or numbered dishes may be used, into each of which ten sections are put. If, then, certain changes are found in any particular location, they may be followed in the sections immediately preceding or succeeding. The eyeball should be cut entirely at one sitting, if possible, as an inexact levelling at a subsequent occasion may result in the loss of valuable material. The sections are now ready to stain.

Small pieces of tissue, such as tumors, portions of the optic nerve, etc., may be mounted much more quickly. From the fixing fluid they are transferred for twenty-four hours each into 75 per cent. alcohol, 95 per cent. alcohol, absolute alcohol, alcohol-ether, thin celloidin, thick celloidin, and then are mounted on a block of wood and covered with a layer of thick celloidin. When this has hardened slightly on the surface (after a few minutes) the blocks are placed in 80 per cent. alcohol for at least six hours, and the material is ready to cut with the microtome.

Paraffin Embedding. The tissue is hardened in the same way as for celloidin, and after twenty-four hours in absolute alcohol it is placed in a mixture of absolute alcohol and chloroform for twenty-four hours, then into pure chloroform for twenty-four hours, chloroform saturated with paraffin (warm) for twenty-four hours, and then melted paraffin in a paraffin oven. Paraffin of two melting points may be used—the first melting at 42°, in which the tissue remains two hours, and the second at 54° to 58°, in which it remains for the same length of time. The oven should be regulated by a thermostat to remain steadily at a temperature slightly above the higher melting point. The chloroform may be replaced by xylol, and for strips of the retina the process may be much shortened (see page 678). Small objects remain in xylol four hours, xylol paraffin six hours, and paraffin up to five hours. The tissue may then be mounted on a block of wood and covered with the melted paraffin by means of a warm spatula, and then thrown into water to harden quickly, or else placed in a shallow glass dish (the walls of which have been coated with glycerin), and covered with paraffin. Small paper boxes may also be made and used for this purpose. As soon as the surface of the paraffin has hardened slightly the entire dish or box is submerged in cold water, in order that the paraffin may harden quickly and evenly. After the superfluous paraffin has been cut away, the block is mounted on a piece of wood by warming the surface of the paraffin, and it is now ready for cutting. In cutting, the knife is not placed at so sharp an angle, and the sections are cut dry. They must be kept from rolling up by means of a fine camel's-hair brush, and should be spread on warm water, in which they flatten out smoothly. If ribbon sections are desired, the block

of paraffin should be cut accurately quadrilateral, and the knife placed at right angles to the microtome. The sections should be stained in a slide, and the paraffin dissolved out before the stain is applied. Many methods are employed to fasten them to the slide so that they shall not be floated away. The slides, in the first place, should be cleaned scrupulously with alcohol and dipped beneath the section, so that the latter may be floated on. If there is no hurry, the simplest means of cementing them fast is to place the slides on the top of the paraffin oven for twenty-four hours. The paraffin is then dissolved by xylol, the xylol removed by absolute alcohol, and the sections are ready to stain. If aqueous stains are to be used, the slides should be placed in 80 per cent. alcohol, and then in water. If immediate staining is necessary, the sections may be pressed firmly to the slide by means of filter-paper moistened with absolute alcohol, the paraffin is dissolved with xylol, the xylol removed by absolute alcohol, and the sections covered with a *very thin* solution of celloidin. When this thin layer hardens the slide is placed in 80 per cent. alcohol, and then in water. The celloidin does not interfere with the subsequent staining, and the sections remain in position.

Staining Methods. Before microscopic study the sections should be stained. For this purpose we make use of two types of stains: 1. Those which stain electively the nuclei; and, 2. Those which stain diffusely the cell protoplasm. Of the former, we may confine ourselves practically to two—hæmatoxylin and carmine; and of the latter, eosin, fuchsin, and picric acid are especially valuable. Staining in bulk rarely is used, and each section should be handled separately. After suitable staining the sections are dehydrated in alcohol, cleared in one of the essential oils, or mixture of carbolic acid and xylol, and mounted permanently in Canada balsam. Staining with hæmatoxylin and counterstaining with eosin may be described briefly as follows:

1. The sections are placed in water to remove the alcohol, and then into Delafield's hæmatoxylin. A well-ripened solution is added to filtered tap-water until a layer one half an inch deep can just be seen through. In this they remain three to five minutes, until sufficiently stained (the celloidin should be colored light blue). If, on removal to tap-water, the stain is not sufficiently deep, replace in the staining solution. It is better to overstain than to understain.

2. Wash thoroughly in filtered tap-water, to which a drop or two of ammonia water may be added, if it is not sufficiently alkaline to give the sections a deep-blue color. If the sections are overstained, they may be placed in a 0.5 per cent. solution of hydrochloric acid in 70 per cent. alcohol a moment, until the celloidin loses most of its color. Then wash thoroughly with alkaline tap-water.

3. Distilled water.

4. Thin alcoholic solution of eosin, one minute.

5. 95 per cent. alcohol, to remove excess of eosin and to dehydrate. Carefully straighten out the sections on the spatula, and float them on the surface of

6. Carbol-xylol (xylol 3, carbolic acid crystals 1), where they should spread out smoothly. Here all remaining traces of water are removed, and the sections should show no white patches in the tissue when held over a dark surface.

7. Transfer the section by means of the spatula to the slide, smooth it out, and press it firmly to the slide with a fine filter-paper folded in six to eight thicknesses. A drop of xylol balsam is dropped on and a cover-glass carefully lowered upon it. All air bubbles should be removed by gentle pressure with the needle, and the section is permanently mounted. Too much balsam is preferable to too little, as the specimen may be spoiled later by the appearance of air bubbles as the balsam dries.

Instead of carbol-xylol, oil of bergamot, origanum, or cajeput may be used for clearing, but the section should be passed through absolute alcohol, and more skilful handling is required, as too long action of the absolute alcohol softens the celloidin.

Van Gieson's Method. 1. The sections are stained with hæmatoxylin, as before, and should be overstained. No differentiation with acid is required.

2. Water.

3. Van Gieson's solution (concentrated aqueous solution of picric acid, to which acid fuchsin is added until a red color of the desired depth is obtained), thirty seconds.

4. Water, for a moment.

5. 95 per cent. alcohol to dehydrate.

6. Absolute alcohol.

7. Xylol.

8. Balsam.

The nuclei are stained brownish red, the other substances deep red to yellow. Axis-cylinders are red, and the nerve sheaths yellow. Muscle tissue is yellow, while connective-tissue fibres are red.

Paraffin sections are stained in the same way, except that they are stained on the slides, and the staining usually requires a longer time. Staining dishes with arrangements for holding the slides apart are great time-savers, as a number of slides may be handled at the same time.

Carmine Stains. These are especially valuable when the sections are to be stained for micro-organisms or fibrin, or when the reaction for iron is to be applied. A number of formulas are used, but the two following methods will suffice :

1. Lithium carmine (2.5 gr. of carmine are dissolved in 100 c.c. of a cold saturated solution of lithium carbonate). Stain for ten minutes. Differentiate in acid alcohol (1 per cent. sol. of HCl in 70 per cent. alcohol) for fifteen minutes, wash in water, dehydrate in alcohol, xylol, balsam.

2. Borax carmine (carmine 5 gm. and borax 2 gm., are dissolved in 100 c.c. of water, the solution is boiled, and 5 c.c. of a 0.5 per cent. solution of acetic acid is added; filter after twenty-four hours).

Stain for fifteen minutes, wash in water, differentiate in acid alcohol fifteen minutes, wash in water, alcohol, xylol, balsam.

Double stains with carmine may be obtained by adding to 1 part of the lithium carmine solution 2 parts of a saturated picric acid solution. The nuclei will be stained red and the remaining protoplasm yellow.

Special Staining Methods. I. WEIGERT'S STAIN FOR NERVE SHEATHS.

1. Fix in Müller's solution, and harden subsequently in alcohol, *without washing the tissue.*

2. Embed in celloidin. Eighty per cent. alcohol, several hours.

3. Saturated solution of neutral acetate of copper, diluted one-half, in the incubator at 35° C., twenty-four hours.

4. Wash. Place in 70 per cent. alcohol six to twelve hours. Cut.

5. Stain in Weigert's alcohol hæmatoxylin (1 gm. of hæmatoxylin is added to 10 c.c. of absolute alcohol and 100 c.c. of water, and the solution boiled; add to this solution a saturated solution of lithium carbonate in the proportion of 1: 100 at the time of using). The stain should be used cold, and may be allowed to act twelve to twenty-fours, the sections staining an intense black.

6. Wash thoroughly in water.

7. Differentiate in a solution composed of: borax, 2; potassium ferricyanide, 2.5; water, 100.

The normal nerve sheaths retain the black color, while the degenerated fibres and the remaining tissue become light brown. If the differentiation proceeds too rapidly, the solution should be diluted. The process should be interrupted from time to time and the sections examined under the microscope, as the optic nerve fibres are exceedingly fine in calibre and are decolorized much more quickly than those of the central nervous system. The right time for interrupting it may be judged by watching the ciliary nerves, which often are included in the section.

8. Wash thoroughly in water until all traces of the differentiating solution are removed, dehydrate in alcohol, carbol-xylol, balsam.

This method is readily applied if the optic nerve has been cut far enough back of the eye to allow the removal of a piece for special examination. Good results may also be obtained without coppering the pieces if the sections in celloidin are placed for twenty-four hours in 0.5 per cent. solution of chromic acid in the incubator. They are then stained and differentiated as before, but should be watched carefully. The fibres will be stained bluish black rather than dead black. This method is applicable to the sections of the eyeball, provided that the eye has been hardened in Müller's fluid. Sections fixed in formalin alone will not give certain results, even if they are treated with the chromic acid solution, and if the examination of the nerve fibres is of importance, Müller's fluid should always be used for fixing. Where this would interfere with an examination of the retina, a small strip may be removed as described in speaking of the methods or studying the ganglion cells.

II. Marchi's Method. 1. Fix small pieces of the optic nerve in Müller's fluid, eight days.

2. Freshly prepared mixture of Müller's fluid and 1 per cent. osmic acid solution in equal parts. Six to twelve days.

3. Wash in running water. Twenty-four hours.

4. Alcohol, celloidin. Cut. The sections are dehydrated in alcohol, cleared in carbol-xylol, and mounted in Canada balsam. The degenerated nerve fibres appear as fine black dots arranged in chains; fat tissue around the nerve is also stained black. All else is light yellow, often with a greenish tinge. The sections may also be stained with carmine, Van Gieson's fluid, etc. If permanent preparations are desired, the sections should not be covered with a cover-glass, otherwise the black color is apt to fade quickly. On the cut surface of the nerve there is always a deposit of black dots, but they do not extend far into the substance of the nerve.

III. Ganglion Cells of the Retina. The study of the ganglion cells of the retina has become of great importance, especially in connection with various intoxications. Sections made through the eyeball, in celloidin, are usually too thick for this purpose, and small strips of the retina may be cut out with a sharp pair of scissors when the eye is cut in half. If Müller's fluid is to be used, a cut should be made with a sharp instrument through sclera, choroid, and retina immediately after enucleation, the retina carefully lifted from the underlying choroid, and a strip excised. It may be placed in 20 per cent. formalin, or in 96 per cent. alcohol for twenty-four hours, then into absolute alcohol one hour, xylol for one hour, xylol paraffin (concentrated solution of paraffin in warm xylol) one hour, and, finally, for fifteen minutes each in soft and hard paraffin. The sections should be from 2 to 6 μ thick.

a. Staining with Thionin. 1. 10 per cent. aqueous solution of thionin ten minutes.

2. Wash rapidly in water.

3. Differentiate in 96 per cent. alcohol (watch under the microscope).

4. Absolute alcohol, xylol, balsam.

The sections may also be stained with a concentrated aqueous solution of thionin, and subsequently differentiated with aniline oil, 1 gm.; absolute alcohol, 9 gm.; cleared with xylol, and mounted in balsam. The Nissl bodies in the protoplasm surrounding the nucleus of the ganglion cell are stained deep blue, the nuclei a paler blue. Contrast-stains with eosin or erythrosin may be used, but are apt to blur the finer details.

b. Staining with Toluidine-blue (Hoyer and V. Lenhossek).

1. Fix in concentrated corrosive sublimate solution, twenty-four hours.

2. Harden in alcohol.

3. Embed in paraffin (using chloroform as a solvent).

4. Cut. Mount with distilled water. Extract paraffin with xyol and iodine-alcohol (solution of iodine in absolute alcohol).

5. Stain with concentrated aqueous solution of toluidine-blue several hours.

6. Differentiate in aniline-alcohol. Counterstain with alcoholic eosin solution (or erythrosin).

7. Rapidly dehydrate in absolute alcohol, xylol, balsam.

The stains are not usually permanent.

IV. STAIN FOR NEUROGLIA. The determination of the condition of the neuroglia may be of value in the study of the optic nerve. Weigert's method is a long and rather difficult one, and good results may be obtained by either of Mallory's methods, especially when the neuroglia is pathologically increased. The first method is as follows:

1. Fix in formalin (10 per cent.) four days.
2. Concentrated aqueous solution of picric acid, four to eight days.
3. 5 per cent. solution of ammonium bichromate. Four to six days in the incubator at 37°. Change the solution on the second day.
4. Alcohol.
5. Celloidin.
6. Stain by Weigert's fibrin method (see below).
7. Differentiate with aniline oil and xylol (of each, equal parts), xylol, balsam. As contrast-stain, fuchsin may be added to the aniline oil.

The second method is said to be especially suited for demonstrating the neuroglia in glioma of the retina. The tissue is treated as in the first method until the celloidin sections are cut. They are then placed in

1. 0.5 per cent. aqueous solution of potassium permanganate twenty-five to thirty minutes.
2. Wash in water.
3. 1 per cent. aqueous solution of oxalic acid fifteen to thirty minutes.
4. Wash in two or three changes of water.
5. Stain in phosphotungstic acid hæmatoxylin one to three days. The formula of this is: hæmatoxylin, 0.1 gm.; water, 80 gm.; phosphotungstic acid (Merck), 20 gm.; peroxide of hydrogen, 0.2 gm. Dissolve the hæmatoxylin in a little water, by aid of heat, and add it, after cooling, to the rest of the solution.
6. Wash quickly in water.
7. Dehydrate in 95 per cent. alcohol.
8. Oleum origani cretici.
9. Xylol balsam.

The nuclei, neuroglia fibres, and fibrin stain blue, axis-cylinders and ganglion cells pale pink, connective tissue deep pink. The blue color is slightly sensitive to light, and is apt to fade to pink after prolonged exposure. If a permanent isolated stain of the neuroglia fibres is desired, transfer the sections (after staining in the phosphotungstic acid hæmatoxylin and washing in water) to a 30 per cent. alcoholic solution of ferric chloride for five to twenty minutes, then wash in water, and dehydrate as before. The nuclei, neuroglia fibres,

and fibrin stand out sharply of a clear blue color. Everything else is decolorized, or appears of a pale yellowish or grayish tint.

Staining for Bacteria in Sections. For this purpose the sections should be as thin as possible, and the eyeball should not be cut as a whole, but divided after embedding in celloidin. With other tissues paraffin should be used as the embedding substance. The use of Müller's fluid for fixation makes the search very difficult, as the organisms do not stain well. If, however, the sections are placed for several hours in a 5 per cent. solution of oxalic acid, satisfactory results may be obtained.

Staining Methods. I. *Methylene-blue.* 1. Stain in Loeffler's alkaline methylene-blue ten minutes (concentrated alcoholic methylene-blue solution 30 gm.; caustic potash solution (1:10,000), 100 gm.).

2. Wash in water.

3. Differentiate in 0.5 per cent. acetic acid one to three seconds.

4. Water.

5. 95 per cent. alcohol, absolute alcohol, bergamot oil, and xylol balsam.

The organisms and cell nuclei are stained blue. If the bacteria retain their color by the Gram method, this may be employed. It is a valuable stain, as it reveals the presence of fibrin at the same time.

II. *Gram-Weigert Method.* 1. The sections may be stained first with lithium carmine (see page 676) for contrast. After washing in water the sections should be spread out carefully on a well-cleaned slide, so that no wrinkles appear, and pressed firmly to the glass by filter-paper in layers. The stain should be freshly prepared, and may be made by shaking 0.5 c.c. of transparent aniline oil, with 5 c.c. of water in a test-tube, and filtering through a fine filter-paper moistened with water. To this is added a filtered, concentrated alcoholic solution of gentian or methyl-violet, in the proportion of 1:10. Stain for three to five minutes, pour off the solution, and dry the sections carefully with the filter-paper.

2. Lugol's solution (iodine, 1 gm.; potassium iodide, 2 gm.; water 100 gm.), two minutes. Dry *thoroughly.*

3. Differentiate with aniline oil–xylol (2 : 1) until no further color is given off.

4. Remove aniline oil thoroughly with xylol.

5. Xylol balsam.

The organisms and the fibrin will be stained a deep violet; certain hyaline substances, horny cells, karyokinetic figures, and mucus are also stained by the method. The other nuclei should be stained red by the carmine. Paraffin sections are stained in the same way, but the drying should be done very carefully.

III. *Staining of Tubercle Bacillus in Tissue.* 1. Stain in Ziehl's carbol-fuchsin solution for two hours in the incubator, or in cold solution for twenty-four hours. (Fuchsin, 1 gm.; absolute alcohol, 10 gm.; 5 per cent. carbolic acid solution, 100 gm.)

2. Wash in water.
3. 15 per cent. solution of nitric acid for a few seconds. The section turns brown.
4. Wash thoroughly in water.
5. 95 per cent. alcohol until the section is rose red.
6. Water.
7. Aqueous solution of methylene-blue, one-half minute.
8. Water.
9. Dehydrate in alcohol.
10. Oil of bergamot. Balsam. (Carbol-xylol should never be used for clearing tissue stained in an aniline dye.)

The methods of staining which have been given include the most important that are used for the determination of pathological changes. A description of special methods for histological structure, such as the Golgi methods for the retina, Ehrlich's methylene-blue stain for living tissue, silver and gold methods for the cornea, etc., would exceed the limits of the present article, and the student who desires to pursue advanced researches on such lines is referred to special books on technique.[1]

Bacteriological Examinations. Descriptions of the Organisms that are Most Frequently Found in Diseases of the Conjunctiva and Cornea. Bacteriological studies are of value only in the acute stages of corneal and conjunctival inflammations. The cover-slip examination may then be conclusive, by reason of the large numbers of the organism which are present. Later the specific organism may be crowded out by ordinary saprophytic germs, which are readily introduced into the open conjunctival sac. Cultivation of the organism is, in many cases, very important, but this is very difficult with some of the special forms, as they are readily masked by others which are accidentally present. If possible, where the discharge is abundant, the eye should be washed out and the patient allowed to wait ten or fifteen minutes. A portion of the reaccumulated discharge is then picked up by means of a sterile platinum loop, and smeared on the surface of a carefully cleaned cover-glass or slide. After drying in the air, the smear is passed three times through the flame of a Bunsen burner, and then stained with solution of an aniline dye. Loeffler's alkaline methylene-blue (page 680) is one of the most useful. It stains deeply in five to ten minutes. The cover-glass is then washed in water, dried, mounted in Canada balsam, and examined with a one-twelfth oil-immersion lens. The appearance of several of the special conjunctival organisms, notably the Koch-Weeks bacillus, and the diplobacillus of Morax-Axenfeld, is sufficiently characteristic to allow a positive diagnosis by means of the cover-glass examination. A second smear should be stained by the Gram-Weigert method, as follows:

[1] Two excellent monographs have recently appeared in German: "Anleitung zur mikroskopischen Untersuchung des Auges," by Professor R. Greeff, and "Die mikroskopischen Untersuchungsmethoden des Auges," by S. Seligmann, the latter of which contains these and other special methods in detail.

1. Aniline water, gentian-violet solution (page 680), three to five minutes.
2. Lugol's solution, two minutes.
3. 95 per cent. alcohol until no further stain is removed.
4. Water.
5. Counterstain with aqueous fuchsin *not longer than twenty to thirty seconds.* Certain of the organisms retain the deep violet color, and are termed *positive;* others lose the stain and are colored red by the fuchsin, and are termed *negative* organisms.

If the cover-slip examination is not conclusive, cultures should be made. For this purpose coagulated blood serum, glycerin agar, and weak agar are especially adapted. Great care should be taken to prevent contamination by other organisms which are present along the lid margins, and, as in the cover-slip examination, the eye should first be washed out with distilled water and the discharge allowed to reaccumulate. A small portion is then picked up by a platinum loop, and the surfaces of several slant-tube cultures or Petri dishes are inoculated. Two days' growth in the incubator (37° to 39°) will usually show the presence of characteristic colonies.

In obtaining material from the cornea the eyes should be washed out with sterile water, a sterilized cocaine solution instilled, and while the eyelids are carefully held away from the cornea a portion of the material in the bed of the ulcer is removed by means of a pointed, sterilized lance, and transferred to the media. For the details of this work and those of animal inoculation, text-books on bacteriological technique should be consulted. The most important organisms which are pathogenic for the human conjunctiva are as follows:

1. The Koch-Weeks bacillus.
2. The gonococcus of Neisser.
3. The diplobacillus of Morax-Axenfeld.
4. The diplococcus lanceolatus of Fraenkel-Weichselbaum.
5. The Klebs-Loeffler diphtheria bacillus.
6. Staphylococcus pyogenes.
7. Streptococcus pyogenes.
8. The diplococcus of acute follicular catarrh (pseudogonococcus).

The first three are unconditionally pathogenic for the conjunctiva; that is, they are not present in the normal conjunctiva, and when introduced produce a specific and contagious inflammation. The others may be found on the normal conjunctiva, and produce inflammation only under certain conditions of virulence, lowered vitality of the individual, or local lesion of the conjunctiva, such as chronic inflammation or injury of the surface. Besides these organisms many others, for example, bacterium coli, bacillus of rhinoscleroma, Friedländer's pneumobacillus, the ozæna bacillus and certain of the higher fungi (actinomyces and aspergillus) have been found in isolated cases. The tubercle and lepra bacilli are present in the nodular or ulcerative lesions of the external coats of the eye, and may

be detected in a histological examination of the tissue, but do not cause a conjunctival catarrh in the ordinary sense of the term. The so-called xerosis bacillus is also a frequent occupant of the conjunctival sac both in health and in disease, but cannot be said to be pathogenic.

1. **The Koch-Weeks bacillus,** described first by Koch in Egypt, more thoroughly studied by Weeks in New York, and later by Morax in Paris, and Müller in Vienna. It is the cause of acute contagious conjunctivitis in a varying proportion of *epidemic* cases, depending upon the locality. It is a very small, rod-shaped organism, resembling the bacillus of mouse septicæmia, found in large numbers both within and between the cells of the discharge. Stains readily with methylene-blue or gentian-violet; *negative* to Gram. Usually found mixed with the xerosis bacillus, from which it is difficult to separate in cultures. Culture difficult; best on 0.5 per cent. agar (Weeks), as small punctate, transparent colonies. According to Müller, it grows on human serum agar; only, however, in presence of a certain saprophyte. Unconditionally pathogenic for the human conjunctiva.

2. **The Gonococcus** (Neisser) occurs usually in the form of a diplococcus, the edges in contact being slightly concave, so that the individuals are shaped like a coffee-bean. The organisms are arranged usually in small, irregular groups on and in the cells. Stains readily with methylene-blue; *negative* to Gram. Cultivation is difficult; best on serum covered with human blood. Produces severe purulent ophthalmia, and is the most frequent cause of ophthalmia neonatorum. Unconditionally pathogenic.

3. **Diplobacillus of Morax-Axenfeld.** Large bacillus, measuring on an average 2 μ long and 1 μ broad, with rounded extremities, occurs chiefly in pairs, occasionally in chains, usually free in the secretion in large numbers. Stains readily with aniline dyes; *negative* to Gram. Easily distinguished from Koch-Weeks bacillus by its size. Growth abundant in blood serum in the form of small transparent colonies, which gradually sink below the surface because of their *liquefaction of the serum*. Unconditionally pathogenic for man, producing a subacute catarrh, which yields quickly to solutions of zinc.

4. **Diplococcus Lanceolatus (Pneumococcus) of Fraenkel-Weichselbaum.** Occurs in pairs, the individuals being slightly oval, with pointed outer extremities; occasionally is found in short chains. Each pair may be surrounded by a definite capsule, which, however, is often absent on the conjunctiva. Stains readily with aniline dyes, and is *positive* to Gram. Grows best in glycerin-agar (the media should be slightly alkaline), as delicate transparent colonies, resembling dewdrops. Found in the normal conjunctiva, but may be the cause of acute contagious conjunctivitis, usually of a mild type, with moderate secretion and much fibrin. It is also the cause of a certain proportion of cases of ophthalmia neonatorum, and is the specific organism for a large percentage of cases of ulcus corneæ serpens. Occasionally it is the cause of panophthalmitis.

5. **Klebs-Loeffler Diphtheria Bacillus.** The diphtheria bacillus is exceedingly variable in form, sometimes appearing as straight or slightly curved rods, with pointed ends; at other times spindle and club shapes occur, in which segments less deeply stained appear. On cultures the morphology is even more irregular. It stains best with Loeffler's methylene-blue; grows readily on all ordinary media; best upon Loeffler's blood serum and upon glycerin-agar. It is pathogenic for animals, and causes death with characteristic lesions. It is the cause of diphtheritic conjunctivitis, but is found also in the more superficial (croupous) form, as well as in simple catarrh and on the normal conjunctiva. Morphologically and culturally it may be confounded with the *xerosis bacillus*, a very frequent and harmless occupant of the conjunctiva, both alone and associated with other specific organisms. In cultures, however, the xerosis bacillus does not grow so luxuriantly, the colonies are usually dryer on the surface, the individuals do not show such bizarre forms on blood serum, the so-called Ernst's granules do not appear so soon, and the organism is not pathogenic for animals.

6. **Staphylococcus Pyogenes Aureus.** The staphylococcus pyogenes is one of the most common pus organisms. It occurs as a small spherical coccus, usually in groups and extracellular. Stains by ordinary aniline dyes, and is *positive* to Gram. Grows well on all ordinary culture-media, and can be differentiated only by this method. It is present on the normal conjunctiva in inflammations of the lid margin, and in many forms of ulcer of the cornea (aside from ulcus serpens); it may be associated with other more pathogenic forms, and is found occasionally in pure culture in simple catarrhs and in pseudomembranous conjunctivitis. It is frequently found in the pus of dacryocystitis and in panophthalmitis, both by metastasis and by direct infection.

7. **Streptococcus Pyogenes.** The streptococcus is likewise a spherical coccus, usually slightly larger than the staphylococcus, which occurs in chains of varying length as a result of division in only one direction. It stains readily, and is *positive* to Gram. Grows on artificial media, but less luxuriantly than the staphylococcus. It is found in the normal conjunctiva and frequently in inflammations of the tear ducts. It may be associated with other organisms and increase the severity of the process. It may be the sole cause of conjunctivitis in one of two forms: 1. Catarrhal inflammation (Parinaud's conjunctivitis), which is usually monolateral, and is associated with lacrymal disease of the same side; it is often complicated by iritis and swelling of the preauricular glands. 2. A pseudomembranous form, which is more frequent. Here it may be found alone or with the diphtheria organism. The process is usually very severe and the prognosis bad.

8. **Diplococcus of Acute Follicular Catarrh (Pseudogonococcus).** This organism has been described by many as the cause of acute follicular catarrh in epidemic form. It has a very close resemblance to the

gonococcus on the cover-slip, but is *positive to Gram and is readily cultivated.* The diagnosis of gonococcus should not be made without the use of the Gram method unless the direct source of infection is known. Other forms have been described which were also negative to Gram, but they could be cultivated without difficulty. The meningococcus (diplococcus intracellularis meningitidis, Weichselbaum) might be confounded with the gonococcus, but its occurrence on the conjunctiva is exceptional.

In keratitis a variety of organisms has been cultivated from the ulcers, but in many cases the infection must be looked upon as secondary. Only two forms of keratitis can be considered to be caused by specific micro-organisms: 1. The true ulcus serpens, in which the pneumococcus was found in a large percentage of cases by Uhthoff and Axenfeld. 2. Keratomycosis aspergillinia, a form of ulcer produced by the Aspergillus fumigatus. This form is, however, exceedingly rare in America; only two cases have been reported. Other organisms found have been staphylococci, Pfeiffer's capsule bacillus, bacillus pyogenes fœtidus, bacterium coli, bacillus pyocyaneus, diplobacillus, ozæna bacillus, and a number of other forms which have not been identified (Uhthoff).

INDEX.

Catalogue of Books

PUBLISHED BY

Lea Brothers & Company,

706, 708 & 710 Sansom St., Philadelphia.
111 Fifth Avenue, New York.

Our publications can be purchased from any Bookseller in the United States or Canada, or they will be delivered by express or mail, carriage paid to any address on receipt of the printed price.

JULY, 1903.

STANDARD MEDICAL PERIODICALS.

Progressive Medicine.

A Quarterly Digest of New Methods, Discoveries and Improvements in the Medical and Surgical Sciences by Eminent Authorities. Edited by DR. HOBART AMORY HARE. In four abundantly illustrated, cloth bound, octavo volumes of 400–500 pages each, issued quarterly, commencing with March each year. Per annum (4 volumes), $10.00, delivered.

The Medical News.

WEEKLY, $4.00 PER ANNUM.

Each number contains 48 quarto pages, abundantly illustrated. A crisp, fresh weekly professional newspaper.

The American Journal of the Medical Sciences.

MONTHLY, $5.00 PER ANNUM.

Each issue contains 192 octavo pages, fully illustrated. The most advanced, practical, original and enterprising American exponent of scientific medicine.

The Medical News Visiting List for 1903.

Four styles, Weekly (dated for 30 patients); Monthly (undated, for 120 patients per month); Perpetual (undated, for 30 patients weekly per year); and Perpetual (undated, for 60 patients weekly per year). Each style in one wallet-shaped book, leather bound, with pocket, pencil and rubber. Price, each, $1.25. Thumb-letter index, 25 cents extra.

The Medical News Pocket Formulary.

New (5th) edition carefully revised to date. Containing 1700 prescriptions representing the latest and most approved methods of administering remedial agents. Strongly bound in leather, with pocket and pencil. Price, $1.50, *net.*

COMBINATION RATES.

Per Annum	ALONE.	IN COMBINATION	
American Journal of the Medical Sciences	**$5.00**	**$8.00**	**$16.00**
Medical News	**4.00**		
Progressive Medicine	**10.00**		
Medical News Visiting List	**1.25**		
Medical News Formulary	**1.50, net.**		

In all $21.75 for $17.00

First four above publications in combination......$16.75
All above publications in combination.................. 17.00

Other Combinations will be quoted on request. Full Circulars and Specimens free.

ABBOTT (A. C.). *PRINCIPLES OF BACTERIOLOGY:* a Practical Manual for Students and Physicians. Sixth edition enlarged and thoroughly revised. 12mo., 636 pages, with 111 engravings, of which 26 are colored. Cloth, $2.75, *net.*

ALLEN (CHARLES W.). *A TREATISE ON RADIOTHERAPY AND PHOTOTHERAPY.* Octavo, about 500 pages, with many illustrations. *Shortly.*

ALLEN (HARRISON). *A SYSTEM OF HUMAN ANATOMY.* Comprising 813 quarto pages, with 380 engravings on stone and 241 woodcuts. Cloth, $23.

AMERICAN SYSTEM OF PRACTICAL MEDICINE. In Contributions by Eminent American Authors. Edited by Alfred L. Loomis, M.D., LL.D., and W. Gilman Thompson, M.D. In four octavo volumes of about 900 pages each, fully illustrated. Per volume, cloth, $5; leather, $6; half morocco, $7. *For sale by subscription only.* Prospectus free on application.

AMERICAN SYSTEM OF DENTISTRY. *IN TREATISES BY VARIOUS AUTHORS.* Edited by Wilbur F. Litch, M.D., D.D.S. In three octavo volumes, containing about 3200 pages, with 1873 illustrations and many full-page plates. Per volume, cloth, $6; leather, $7. Prospectus free on application to the Publishers.

AMERICAN TEXT-BOOK OF ANATOMY. See *Gerrish,* page 7.

AMERICAN TEXT-BOOKS OF DENTISTRY. *IN CONTRIBUTIONS BY EMINENT AMERICAN AUTHORITIES.* In two octavo volumes of more than 800 pages each, richly illustrated:

—— *PROSTHETIC DENTISTRY.* Edited by Charles J. Essig, M.D., D.D.S., Professor of Mechanical Dentistry and Metallurgy, Department of Dentistry, University of Pennsylvania, Philadelphia. Second Edition, 807 pages, with 1089 engravings. Cloth, $6; leather, $7, *net.*

—— *OPERATIVE DENTISTRY.* Edited by Edward C. Kirk, D.D.S., Professor of Clinical Dentistry, Department of Dentistry, University of Pennsylvania. Second Edition, 857 pages, 897 engravings. Cloth, $6.00; leather, $7.00, *net.*

AMERICAN SYSTEMS OF GYNECOLOGY AND OBSTETRICS. Gynecology edited by Matthew D. Mann, A.M., M.D., and Obstetrics edited by Barton C. Hirst, M.D. In four large octavo volumes comprising 3612 pages, with 1092 engravings, and 8 colored plates. Per volume, cloth, $5.

ARCHINARD (P. E.). *AN EPITOME OF BACTERIOLOGY.* 12mo., about 240 pages, with many illustrations. Cloth, $1.00, *net. Lea's Series of Medical Epitomes.* See page 10.

ASHHURST (JOHN, JR.). *THE PRINCIPLES AND PRACTICE OF SURGERY.* For the use of Students and Practitioners. Sixth and revised edition. In one 8vo. volume of 1161 pages, with 656 engravings. Cloth, $6; leather, $7.

A SYSTEM OF PRACTICAL MEDICINE BY AMERICAN AUTHORS. Edited by William Pepper, M.D., LL.D. In five large octavo volumes, containing 5573 pages and 198 illustrations. Price per volume, cloth, $5. Prospectus free on application.

A TREATISE ON SURGERY BY AMERICAN AUTHORS. *FOR STUDENTS AND PRACTITIONERS OF SURGERY AND MEDICINE.* Edited by Roswell Park, M.D. Third edition. Octavo, 1408 pages, with 692 engravings and 64 plates. Cloth, $7.00, *net;* leather, $8.00, *net.* ☞ Published also in 2 volumes at $3.75 *net* per vol. for cloth.

ATTFIELD (JOHN). *CHEMISTRY; GENERAL, MEDICAL AND PHARMACEUTICAL.* Sixteenth edition, specially revised by the Author for America. In one handsome 12mo. volume of 784 pages, with 88 illustrations. Cloth, $2.50, *net.*

BACON (GORHAM). *ON THE EAR.* Third Edition. One 12mo. volume, 430 pages, with 120 engravings and 7 colored plates. Cloth, $2.25, *net.*

BALLENGER (W. L.) AND WIPPERN (A. G.). *A POCKET TEXT-BOOK OF DISEASES OF THE EYE, EAR, NOSE AND THROAT.* 12mo., 525 pages, with 148 illustrations, and 6 colored plates. Cloth, $2, *net.* Flexible red leather, $2.50, *net.*

BARNES (ROBERT AND FANCOURT). *A SYSTEM OF OBSTETRIC MEDICINE AND SURGERY.* Octavo, 872 pages, 231 illustrations. Cloth, $5.

BARTHOLOW (ROBERTS). *CHOLERA; ITS CAUSATION, PREVENTION AND TREATMENT.* 12mo., 127 pages, with 9 illustrations. Cloth, $1.25.

BILLINGS (JOHN S.). *THE NATIONAL MEDICAL DICTIONARY.* Including in one alphabet English, French, German, Italian and Latin Technical Terms used in Medicine and the Collateral Sciences. In two imperial octavo volumes, containing 1574 pages and two colored plates. Per vol., leather, $7. Specimen pages on application.

BLACK (D. CAMPBELL). *THE URINE IN HEALTH AND DISEASE, AND URINARY ANALYSIS, PHYSIOLOGICALLY AND PATHOLOGICALLY CONSIDERED* 12mo., 256 pages, 73 engravings. Cloth, $2.75.

BLOODGOOD (JOSEPH C.). *A TREATISE ON THE PRINCIPLES OF SURGERY.* Octavo about 1000 pages, with many engravings and plates in black and colors. *Preparing.*

BLOXAM (C. L.). *CHEMISTRY, INORGANIC AND ORGANIC.* With Experiments. New American from the fifth London edition. In one handsome octavo volume of 727 pages, with 292 illustrations. Cloth, $2; leather, $3.

BREWER (GEORGE E.) *A TEXT-BOOK OF THE PRINCIPLES AND PRACTICE OF SURGERY.* Octavo, 700 pages, with 280 engravings and 7 plates. Cloth, $4.00; leather, $5.00, *net.* *Just Ready.*

BRUCE (J. MITCHELL). *MATERIA MEDICA AND THERAPEUTICS.* Sixth edition. In one 12mo. volume of 600 pages. Cloth, $1.50, *net.*
Students' Series of Manuals. Page 15.

—— *PRINCIPLES OF TREATMENT.* In one octavo volume of 625 pages. Cloth, $3.75, *net.*

BRYANT (THOMAS). *THE PRACTICE OF SURGERY.* Fourth edition. Octavo, 1040 pages, with 727 illustrations. Cloth, $6.50; leather, $7.50.

BURCHARD (HENRY H.). *DENTAL PATHOLOGY AND THERAPEUTICS, INCLUDING PHARMACOLOGY.* Handsome octavo, 575 pages, with 400 illustrations. Cloth, $5; leather, $6, *net.*

BURNETT (CHARLES H.). *A TREATISE ON THE EAR.* Second edition. 8vo., 580 pages, with 107 illustrations. Cloth, $4; leather, $5.

CARTER (R. BRUDENELL) AND FROST (W. ADAMS). *OPHTHALMIC SURGERY.* In one pocket-size 12mo. volume of 559 pages, with 91 engravings and one plate. Cloth, $2.25. See *Series of Clinical Manuals*, page 13.

CASPARI (CHARLES, JR.). *A TREATISE ON PHARMACY.* For Students and Pharmacists. Second edition. Revised and enlarged. Octavo, 774 pages, with 302 illustrations. Cloth, $4.25, *net.*

CHAPMAN (HENRY C.). *HUMAN PHYSIOLOGY.* Second edition. Octavo, 921 pages, with 595 illustrations. Cloth, $4.25; leather, $5.25, *net.*

CHARLES (T. CRANSTOUN). *PHYSIOLOGICAL AND PATHOLOGICAL CHEMISTRY.* Octavo, 451 pages, with 38 engravings and 1 colored plate. Cloth, $3.50.

CHEYNE (W. WATSON) AND BURGHARD (F. F.). *SURGICAL TREATMENT.* In seven octavo cloth bound volumes, illustrated. Volume I, 299 pages and 66 engravings; $3.00, *net.* Volume II, 382 pages, 141 engravings; $4.00, *net.* Volume III, 300 pages, 100 engravings; $3.50, *net.* Volume IV, 383 pages, 138 engravings; $3.75, *net.* Volume V, 482 pages, 145 engravings; $5.00, *net.* Vol. VI, 498 pages, 124 engravings; $5.00, *net.* Volume VII, $5.75, *net. Complete work Just Ready.*

CLELAND (JOHN). *A DIRECTORY FOR THE DISSECTION OF THE HUMAN BODY.* In one 12mo. volume of 178 pages. Cloth, $1.25.

CLOUSTON (THOMAS S.). *CLINICAL LECTURES ON MENTAL DISEASES.* New (5th) edition. Crown 8vo., of 736 pages with 19 colored plates. Cloth, $4.25, *net.* FOLSOM'S *Abstract of Laws of U.S. on Custody of Insane*, octavo, $1.50, is sold in conjunction with *Clouston on Mental Diseases* for $5.00, *net*, for the two works.

COAKLEY (CORNELIUS G.). *DISEASES OF THE NOSE, THROAT, NASOPHARYNX AND TRACHEA.* Second edition. 12mo., 556 pages, with 103 engravings, and 4 colored plates. Cloth, $2.75, *net.*

COATS (JOSEPH). *A TREATISE ON PATHOLOGY.* Octavo, 829 pages, with 339 engravings. Cloth, $5.50; leather, $6.50.

COLEMAN (ALFRED). *A MANUAL OF DENTAL SURGERY AND PATHOLOGY.* Octavo, 412 pages, with 331 engravings. Cloth, $3.25.

COLLINS (H. D.) AND ROCKWELL (W. H., JR.). *A POCKET TEXT-BOOK OF PHYSIOLOGY.* 12mo., of 316 pages, with 153 illustrations. Cloth, $1.50, *net;* flexible leather, $2.00, *net.* *Lea's Series of Pocket Text-Books.* Page 12.

CONDIE (D. FRANCIS). *A PRACTICAL TREATISE ON THE DISEASES OF CHILDREN.* Sixth edition. 8vo. 719 pages. Cloth, $5.25; leather, $6.25.

CORNIL (V.). *SYPHILIS: ITS MORBID ANATOMY, DIAGNOSIS AND TREATMENT.* Translated, by J. HENRY C. SIMES, M.D., and J. WILLIAM WHITE, M.D. 8vo. 461 pages, with 84 illustrations. Cloth, $3.75.

CROCKETT (M. A.). *A POCKET TEXT-BOOK OF DISEASES OF WOMEN.* 12mo. of 368 pages, with 107 illustrations. Cloth, $1.50, *net.* Flexible Red Leather, $2.00, *net.* *Lea's Series of Pocket Text-Books.* Page 12.

CROOK (JAMES K.). *MINERAL WATERS OF UNITED STATES.* Octavo, 574 pages. Cloth, $3.50, *net.*

CULBRETH (DAVID M. R.). *MATERIA MEDICA AND PHARMACOLOGY.* Third edition. Octavo, 905 pages, with 473 engravings. Cloth, $4.75, *net.*

CUSHNY (ARTHUR R.) *PHARMACOLOGY AND THERAPEUTICS.* Third edition. Octavo of 750 pages, with 52 illustrations. Cloth, $3.75; leather, $4.75, *net.*

DALTON (JOHN C.). *A TREATISE ON HUMAN PHYSIOLOGY.* Seventh edition, thoroughly revised. Octavo of 722 pages, with 252 engravings. Cloth, $5; leather, $6.

DALTON (JOHN C.). *DOCTRINES OF THE CIRCULATION OF THE BLOOD.* In one handsome 12mo. volume of 293 pages. Cloth, $2.

DAVENPORT (F. H.). *DISEASES OF WOMEN.* Fourth edition. 12mo., 402 pages and 154 engravings. Cloth, $1.75, *net.*

DAVIS (F. H.). *LECTURES ON CLINICAL MEDICINE.* Second edition. In one 12mo. volume of 287 pages. Cloth, $1.75.

DAVIS (EDWARD P.). *A TREATISE ON OBSTETRICS.* Octavo, 546 pages, with 217 engravings, and 30 full-page plates in colors and monochrome. Cloth, $5; leather, $6.

DE LA BECHE'S *GEOLOGICAL OBSERVER.* In one large octavo volume of 700 pages, with 300 engravings. Cloth, $4.

DENNIS (FREDERIC S.) AND BILLINGS (JOHN S.). *A SYSTEM OF SURGERY.* In Contributions by American Authors. In four very handsome octavo volumes, containing 3652 pages, with 1585 engravings, and 45 full-page plates in colors and monochrome. Per volume, cloth, $6; leather, $7; half morocco, gilt back and top, $8.50. *For sale by subscription only.* Full prospectus free.

DERCUM (FRANCIS X.). Editor. *A TEXT-BOOK ON NERVOUS DISEASES.* By American Authors. Octavo, 1054 pages, with 341 engravings and 7 colored plates.

DE SCHWEINITZ (GEORGE E.). *THE TOXIC AMBLYOPIAS.* Very handsome octavo, 240 pages, 46 engravings, and 9 full-page plates in colors. De luxe binding, $4, *net.*

DRAPER (JOHN C.). *MEDICAL PHYSICS.* A Text-book for Students and Practitioners of Medicine. Octavo of 734 pages, with 376 engravings. Cloth, $4.

DRUITT (ROBERT). *THE PRINCIPLES AND PRACTICE OF MODERN SURGERY.* From the twelfth London edition, edited by STANLEY BOYD, F.R.C.S. Large octavo, 965 pages, with 373 engravings. Cloth, $4; leather, $5.

DUANE (ALEXANDER). *A DICTIONARY OF MEDICINE AND THE ALLIED SCIENCES.* Comprising the Pronunciation, Derivation and Full Explanation of Medical, Dental, Pharmaceutical and Veterinary Terms. Together with much Collateral Descriptive Matter, Numerous Tables, etc. Fourth edition, with Appendix. Square octavo volume of 688 pages with 8 colored plates and thumb index. Cloth, $3.00, *net;* limp leather, $4.00, *net.*

DUDLEY (E. C.). *PRINCIPLES AND PRACTICE OF GYNECOLOGY.* Third edition. Octavo, 756 pages, with 474 engravings, of which 60 are colored, and 22 full page plates in colors and monochrome. Cloth, $5.00, *net;* leather, $6.00, *net;* half morocco, $6.50, *net.*

DUNGLISON (ROBLEY). *A DICTIONARY OF MEDICAL SCIENCE.* Containing a full Explanation of the Various Subjects and Terms of Anatomy, Physiology, Medical Chemistry, Pharmacy, Pharmacology, Therapeutics, Medicine, Hygiene, Dietetics. Pathology, Surgery, Ophthalmology, Otology, Laryngology, Dermatology, Gynecology, Obstetrics, Pediatrics, Medical Jurisprudence, Dentistry, Veterinary Science, etc., etc. By ROBLEY DUNGLISON, M.D., LL.D., late Professor of Institutes of Medicine in the Jefferson Medical College of Philadelphia. Edited by RICHARD J. DUNGLISON, A.M., M.D. Twenty-second edition, thoroughly revised and greatly enlarged and improved, with the Pronunciation, Accentuation and Derivation of the Terms. With Appendix. Imperial octavo of 1350 pages, with thumb letter index. Cloth, $7.00, *net;* leather, $8.00, *net.* *This edition contains portrait of Dr. Robley Dunglison.*

DUNHAM (EDWARD K.). *MORBID AND NORMAL HISTOLOGY.* Octavo, 450 pages, with 360 illustrations. Cloth, $3.25, *net.*

DUNHAM (EDWARD K.). *NORMAL HISTOLOGY.* Second edition. Octavo, 319 pages, with 244 illustrations. Cloth, $2.50, *net.*

DWIGHT (E. W.). *AN EPITOME OF MEDICAL JURISPRUDENCE.* 12mo., about 240 pages. Cloth, $1.00, *net.* *Lea's Series of Medical Epitomes.* See page 10.

—— *AN EPITOME OF TOXICOLOGY.* 12mo., about 225 pages. Cloth, $1.00, *net.* *Lea's Series of Medical Epitomes.* See page 10.

ECKLEY (WILLIAM T.). *A GUIDE TO DISSECTION OF THE HUMAN BODY.* Octavo, 400 pages, with 220 large engravings in black and colors. Cloth, $3.50, *net.*

—— *REGIONAL ANATOMY OF THE HEAD AND NECK FOR STUDENTS AND PRACTITIONERS.* Octavo, 240 pages, with 36 engravings and 20 plates in black and colors. Cloth, $2.50, *net.*

EDES (ROBERT T.). *TEXT-BOOK OF THERAPEUTICS AND MATERIA MEDICA.* In one 8vo. volume of 544 pages. Cloth, $3.50; leather, $4.50.

EDIS (ARTHUR W.). *DISEASES OF WOMEN.* 8vo., 576 pages, with 148 engravings. Cloth, $3; leather, $4.

EGBERT (SENECA). *HYGIENE AND SANITATION.* Second edition. In one 12mo. volume of 427 pages, with 77 illustrations. Cloth, $2.25, *net.*

ELLIS (GEORGE VINER). *DEMONSTRATIONS IN ANATOMY.* Eighth edition. Octavo, 716 pages, with 249 engravings. Cloth, $4.25; leather, $5.25.

EMMET (THOMAS ADDIS). *THE PRINCIPLES AND PRACTICE OF GYNÆCOLOGY.* For the use of Students and Practitioners. Third edition, enlarged and revised. 8vo. of 880 pages, with 150 original engravings. Cloth, $5; leather, $6.

ERICHSEN (JOHN E.). *THE SCIENCE AND ART OF SURGERY.* From the eighth enlarged and revised London edition. In two large octavo volumes containing 2316 pages, with 984 engravings. Cloth, $9; leather, $11.

ESSIG (CHARLES J.). *PROSTHETIC DENTISTRY.* Second Edition. See *American Text-books of Dentistry*, page 2.

ESSIG (C. J.) AND KOENIG (AUGUSTUS). *DENTAL METALLURGY.* Fourth edition. 12mo. 277 pages with 143 engravings. Cloth, $1.75, *net.*

EVANS (DAVID J.). *A POCKET TEXT-BOOK OF OBSTETRICS.* 12mo. of 409 pages, with 148 illustrations. Cloth, $1.75, *net;* limp leather, $2.25, *net.* *Lea's Series of Pocket Text-Books.* Page 12.

EWING (JAMES). *A PRACTICAL TREATISE ON THE BLOOD.* Octavo, 432 pages, with 30 engravings and 14 full-page colored plates. Cloth, $3.50, *net.*

FARQUHARSON (ROBERT). *A GUIDE TO THERAPEUTICS.* Fourth edition, revised by FRANK WOODBURY, M.D. 12mo., 581 pages. Cloth, $2.50.

FIELD (GEORGE P.). *A MANUAL OF DISEASES OF THE EAR.* Fourth edition. Octavo, 391 pages, with 73 engravings and 21 colored plates. Cloth, $3.75.

FINDLEY (PALMER D.). *A TREATISE ON GYNÆCOLOGICAL DIAGNOSIS.* Octavo, 493 pages, 210 engravings, 45 plates in black and colors. Cloth, $4.50; leather, $5.50, *net.* *Just Ready.*

FLINT (AUSTIN). *A TREATISE ON THE PRINCIPLES AND PRACTICE OF MEDICINE.* Seventh edition, thoroughly revised by FREDERICK P. HENRY, M.D. In one large 8vo. volume of 1143 pages, with engravings. Cloth, $5; leather, $6.

FLINT (AUSTIN). *A PRACTICAL TREATISE ON THE HEART.* Second edition, enlarged. Octavo, 550 pages. Cloth, $4.

——— *MEDICAL ESSAYS.* In one 12mo. volume of 210 pages. Cloth, $1.38.

——— *ON THE CHEST AND RESPIRATORY ORGANS.* Second and revised edition. Octavo, 591 pages. Cloth, $4.50.

——— *ON PHTHISIS.* In one 8vo. volume of 442 pages. Cloth, $3.50.

FOLSOM (C. F.). *AN ABSTRACT OF STATUTES OF U. S. ON CUSTODY OF THE INSANE.* In one 8vo. volume of 108 pages. Cloth, $1.50.

FORMULARY, THE NATIONAL. See *National Dispensatory.* Page 14.

FORMULARY, POCKET. Fifth edition. See page 1. $1.50, *net.*

FOSTER (MICHAEL). *A TEXT-BOOK OF PHYSIOLOGY.* Sixth and revised edition. Octavo, 923 pages, with 257 illustrations. Cloth, $4.50; leather, $5.50.

FOTHERGILL (J. MILNER). *HAND-BOOK OF TREATMENT.* Third edition. Octavo, 664 pages. Cloth, $3.75; leather, $4.75.

FOWNES (GEORGE). *A MANUAL OF ELEMENTARY CHEMISTRY.* Twelfth edition. Embodying WATTS' *Physical and Inorganic Chemistry.* Royal 12mo., 1061 pages, with 168 engravings, and 1 colored plate. Cloth, $2.75; leather, $3.25.

FRANKLAND (E.) AND JAPP (F. R.). *INORGANIC CHEMISTRY.* Octavo, 677 pages, with 51 engravings and 2 plates. Cloth, $3.75; leather, $4.75.

FULLER (EUGENE). *DISORDERS OF THE SEXUAL ORGANS IN THE MALE.* Octavo, 238 pages, with 25 engravings and 8 plates. Cloth, $2.

GALLAUDET (BERN B.). *A POCKET TEXT-BOOK OF SURGERY.* 12mo. of about 500 pages, with many illustrations. *In Press.* *Lea's Series of Pocket Text-Books.* Page 12.

GANT (FREDERICK JAMES). *THE STUDENT'S SURGERY.* A Multum in Parvo. In one square octavo volume of 845 pages, with 159 engravings. Cloth, $3.75.

GAYLORD (HARVEY R.) AND ASCHOFF (LUDWIG). *PATHOLOGICAL HISTOLOGY.* With introductory note by WILLIAM H. WELCH, M.D. Quarto, 354 pages, with 81 engravings and 40 full-page plates. Cloth, $7.50, *net.*

GERRISH (FREDERIC H.). *A TEXT-BOOK OF ANATOMY.* By American Authors. Edited by FREDERIC H. GERRISH, M.D. Second edition. Imp. octavo, 937 pages, with 1003 illustrations in black and colors. Cloth, $6.50; sheep, $7.50, *net;* half morocco, $8.00, *net.*

GIBBES (HENEAGE). *PRACTICAL PATHOLOGY AND MORBID HISTOLOGY.* Octavo of 314 pages, with 60 illustrations, mostly photographic. Cloth, $2.75.

GRAY (HENRY). *ANATOMY, DESCRIPTIVE AND SURGICAL.* Fifteenth edition, thoroughly revised. In one imperial octavo volume of 1249 pages, with 780 large and elaborate engravings. Price with illustrations in colors, cloth, $6.25, *net*; leather, $7.25, *net.* Price, with illustrations in black, cloth, $5.50; leather, $6.50, *net.*

GRAYSON (CHARLES P.). *DISEASES OF THE THROAT, NOSE, AND ASSOCIATED AFFECTIONS OF THE EAR.* Octavo, 548 pages, with 129 engravings and 8 plates in colors and monochrome. Cloth, $3.50, *net.*

GREEN (T. HENRY). *PATHOLOGY AND MORBID ANATOMY.* Ninth revised edition. Octavo, 565 pages, with 339 engravings and 4 colored plates. Cloth, $3.25, *net.*

GREENE (WILLIAM H.). *MEDICAL CHEMISTRY.* 12mo., 310 pages, with 74 illustrations. Cloth, $1.75.

GRINDON (JOSEPH). *A POCKET TEXT-BOOK OF SKIN DISEASES.* 12mo. of 367 pages, with 39 illustrations. Cloth, $2.00; flexible leather, $2.50, *net.* *Lea's Series of Pocket Text-Books.* Page 12.

GROSS (SAMUEL D.). *THE URINARY BLADDER, THE PROSTATE GLAND AND THE URETHRA.* Third edition, revised by SAMUEL W. GROSS, M.D. Octavo of 574 pages, with 170 illustrations. Cloth, $4.50.

GUENTHER (A. E. AND T. C.). *AN EPITOME OF PHYSIOLOGY.* 12mo., 225 pages, illustrated. Cloth, $1.00, *net.* *Lea's Series of Medical Epitomes.* See page 10.

HABERSHON (S. O.). *DISEASES OF THE ABDOMEN.* Second American from the third English edition. Octavo, 554 pages, with 11 engravings. Cloth, $3.50.

HALL (WINFIELD S.). *TEXT-BOOK OF PHYSIOLOGY.* Octavo, 672 pages, with 343 engravings and 6 colored plates. Cloth, $4.00, *net;* leather, $5.00, *net.*

HAMILTON (ALLAN McLANE). *NERVOUS DISEASES.* Second and revised edition. Octavo, 598 pages, with 72 engravings. Cloth, $4.

HARDAWAY (W. A.). *MANUAL OF SKIN DISEASES.* Second edition. 12mo., 560 pages with 40 illustrations and 2 colored plates. Cloth, $2.25, *net.*

HARE (HOBART AMORY). *A TEXT-BOOK OF PRACTICAL THERAPEUTICS,* with Special Reference to the Application of Remedial Measures to Disease and their Employment upona Rational Basis. Ninth revised edition. Octavo, 851 pages, 105 engravings, 4 colored plates. Cloth, $4.00, *net;* leather, $5.00, *net;* half morocco, $5.50, *net.*

——— *PRACTICAL DIAGNOSIS.* The Use of Symptoms in the Diagnosis of Disease. Fifth edition, revised and enlarged. Octavo, 727 pages, with 236 engravings, and 25 full-page plates. Cloth, $5.00; leather, $6.00; half morocco, $6.50, *net.*

——— **Editor.** *A SYSTEM OF PRACTICAL THERAPEUTICS.* By American and Foreign Authors. Second edition. In three large octavo volumes containing 2593 pages, with 457 engravings and 26 full-page plates. Price per volume, cloth, $5.00, *net;* leather, $6.00, *net;* half morocco, $7.00, *net.* *For sale by subscription only.* Full prospectus free on application to the publishers.

——— *ON THE MEDICAL COMPLICATIONS AND SEQUELÆ OF TYPHOID FEVER.* Octavo, 276 pages, 21 engravings, and2 full-page plates. Cloth, $2.40, *net.*

HARRINGTON (CHARLES). *A TREATISE ON PRACTICAL HYGIENE.* Second edition. Handsome octavo of 755 pages, with 113 engravings and 12 plates in colors and monochrome. Cloth, $4.25, *net.*

HARTSHORNE (HENRY). *ANATOMY AND PHYSIOLOGY.* 12mo., 310 pages, with 220 engravings. Cloth, $1.75.

——— *A CONSPECTUS OF THE MEDICAL SCIENCES.* Comprising Manuals of Anatomy, Physiology, Chemistry, Materia Medica, Practice of Medicine, Surgery and Obstetrics. Second edition. 12mo., 1028 pages, with 477 illustrations. Cloth, $4.25; leather, $5.

HAYDEN (JAMES R.). *A POCKET TEXT-BOOK OF VENEREAL DISEASES.* Third edition. In one 12mo. volume of 304 pages, with 66 engravings. Cloth, $1.75, *net.*; Flexible red leather, $2.25, *net.* *Lea's Series of Pocket Text-Books.* Page 12.

HAYEM (GEORGES) AND HARE (H. A.). *PHYSICAL AND NATURAL THERAPEUTICS.* Heat, Electricity, Climate and Mineral Waters. Edited by H. A. HARE, M.D. Octavo, 414 pages, with 113 engravings. Cloth, $3.

HERMAN (G. ERNEST). *FIRST LINES IN MIDWIFERY.* 12mo., 198 pages, with 80 engravings. Cloth, $1.25. See *Students' Series of Manuals.* Page 15.

HERRICK (JAMES B.). *A HANDBOOK OF DIAGNOSIS.* In one handsome 12mo. volume of 429 pages, with 80 engravings and 2 colored plates. Cloth, $2.50.

HERTER (C. A.). *LECTURES ON CHEMICAL PATHOLOGY.* In one 12 mo. volume of 454 pages. Cloth, $1.75, *net.*

HILL (BERKELEY). *SYPHILIS AND LOCAL CONTAGIOUS DISORDERS.* In one 8vo. volume of 479 pages. Cloth, $3.25.

HILLIER (THOMAS). *A HANDBOOK OF SKIN DISEASES.* Second edition. In one royal 12mo. volume of 353 pages, with two plates. Cloth, $2.25.

HIRST (BARTON C.) AND PIERSOL (GEORGE A.). *HUMAN MONSTROSITIES.* Magnificent folio, containing 220 pages of text and illustrated with 123 engravings and 39 large photographic plates from nature. In four parts, price each, $5.

HOBLYN (RICHARD D.). *A DICTIONARY OF THE TERMS USED IN MEDICINE AND THE COLLATERAL SCIENCES.* Thirteenth edition. In one 12mo. volume of 845 pages. Cloth, $3.00, *net.*

HOLMES (TIMOTHY). *A TREATISE ON SURGERY.* Fifth edition. Octavo, 1008 pages, with 428 engravings. Cloth, $6; leather, $7.

——— *A SYSTEM OF SURGERY.* Edited by John H. Packard, M.D. In three 8vo. volumes, 3137 pages, 979 engravings, 13 plates. Per volume, Cloth, $6.

HUNTINGTON (GEORGE S.). *ABDOMINAL ANATOMY.* Imperial quarto, 590 pages, including 300 full-page plates. De luxe edition, $10.00, *net.*

HYDE (JAMES NEVINS) AND MONTGOMERY (FRANK H.). *A PRACTICAL TREATISE ON DISEASES OF THE SKIN.* Sixth edition, thoroughly revised. Octavo, 832 pages, with 107 engravings and 27 full-page plates, 9 of which are colored. Cloth, $4.50, *net;* leather, $5.50, *net;* half morocco, $6.00, *net.*

JACKSON (GEORGE THOMAS). *THE READY-REFERENCE HANDBOOK OF DISEASES OF THE SKIN.* Fourth edition. 12mo. volume of 617 pages, with 82 engravings, and 3 colored plates. Cloth, $2.75, *net.*

JAMIESON (W. ALLAN). *DISEASES OF THE SKIN.* Third edition. Octavo, 656 pages, with 1 engraving and 9 double-page chromo-lithographic plates. Cloth, $6.

JEWETT (CHARLES). *ESSENTIALS OF OBSTETRICS.* Second edition. 12mo., 385 pages, with 80 engravings and 5 colored plates. Cloth, $2.25, *net.*

——— *THE PRACTICE OF OBSTETRICS.* By American Authors. Second edition. One octavo volume of 775 pages, with 445 engravings in black and colors, and 35 full-page colored plates. Cloth, $5.00; leather, $6.00; half morocco, $6.50.

JULER (HENRY). *A HANDBOOK OF OPHTHALMIC SCIENCE AND PRACTICE.* Second edition. Octavo, 549 pages, with 201 engravings, 17 chromo-lithographic plates, test-types and Color-Blindness Test. Cloth, $5.50; leather, $6.50.

KELLY (A. O. J.) *A MANUAL OF THE PRACTICE OF MEDICINE.* Octavo, about 600 pages, illustrated. *Preparing.*

KIRK (EDWARD C.). *OPERATIVE DENTISTRY.* Second edition. See *American Text-books of Dentistry*, page 2.

KING (A. F. A.). *A MANUAL OF OBSTETRICS.* Eighth edition. In one 12mo. volume of 612 pages, with 264 illustrations. Cloth, $2.50, *net.*

KLEIN (E.). *ELEMENTS OF HISTOLOGY.* Fifth edition. In one pocket-size 12mo. volume of 506 pages, with 296 engravings. Cloth, $2.00, *net.* *Students' Series of Manuals.* Page 15.

KOPLIK (HENRY). *DISEASES OF INFANCY AND CHILDHOOD.* Octavo, 675 pages with 169 engravings and 32 plates in black and colors. Cloth, $5.00; leather, $6.00, *net.* *Just Ready.*

KÜSTNER (OTTO) AND BEYEA (HENRY D.). *A TEXT-BOOK OF GYNECOLOGY.* Octavo, 400 pages, with 260 engravings. *Preparing.*

LANDIS (HENRY G.). *THE MANAGEMENT OF LABOR.* In one handsome 12mo. volume of 329 pages, with 28 illustrations. Cloth, $1.75.

LA ROCHE (R.). *YELLOW FEVER.* In two 8vo. volumes of 1468 pages. Cloth, $7.

LEA (HENRY C.). *CHAPTERS FROM THE RELIGIOUS HISTORY OF SPAIN; CENSORSHIP OF THE PRESS; MYSTICS AND ILLUMINATI; THE ENDEMONIADAS; EL SANTO NINO DE LA GUARDIA; BRIANDA DE BARDAXI.* In one 12mo. volume of 522 pages. Cloth, $2.50.

—— *A HISTORY OF AURICULAR CONFESSION AND INDULGENCES IN THE LATIN CHURCH.* In three octavo volumes of about 500 pages each. Per volume, cloth, $3.

—— *THE MORISCOS OF SPAIN: THEIR CONVERSION AND EXPULSION.* In one royal 12mo. volume of about 425 pages. Extra cloth, $2.25, *net.*

—— *STUDIES IN CHURCH HISTORY.* New edition. 12mo., 605 pages. Cloth, $2.50.

—— *SUPERSTITION AND FORCE; ESSAYS ON THE WAGER OF LAW, THE WAGER OF BATTLE, THE ORDEAL AND TORTURE.* Fourth edition, thoroughly revised. In one royal 12mo. volume of 629 pages. Cloth, $2.75.

LEA'S SERIES OF MEDICAL EPITOMES. Covering the entire field of medicine and surgery in twenty convenient volumes of about 250 pages each, amply illustrated and written by prominent teachers and specialists. Compendious, authoritative and modern. Following each chapter is a series of questions which will be found convenient in quizzing. The Series is constituted as follows:

Hale's Anatomy. Guenther's Physiology. McGlannan's Chemistry and Physics. Kiepe's Materia Medica and Therapeutics. Dayton's Practice of Medicine. Hollis's Physical Diagnosis. Arneill's Clinical Diagnosis and Urinalysis. Nagle's Nervous and Mental Diseases. Wathen's Histology. Stenhouse's Pathology. Archinard's Bacteriology. Magee and Johnson's Surgery. Alling, Griffen and Ferguson's Eye, Ear, Nose and Throat. Schmidt's Genito-Urinary and Venereal Diseases. Schalek's Dermatology. Pedersen's Gynæcology. Manton's Obstetrics. Tuley's Pediatrics. Dwight's Jurisprudence. Dwight's Toxicology.

LEA'S SERIES OF POCKET TEXT-BOOKS. See page 12.

LE FEVRE (EGBERT). *A TEXT-BOOK OF PHYSICAL DIAGNOSIS.* 12mo., 450 pages, 74 engravings, 12 plates. Cloth, $2.25, *net.*

LONG (ELI). *DENTAL MATERIA MEDICA AND THERAPEUTICS.* 12mo., 321 pages, 6 engravings, 18 plates. Cloth, $3.00, *net.*

LOOMIS (ALFRED L.) AND THOMPSON (W. GILMAN), Editors. *A SYSTEM OF PRACTICAL MEDICINE.* In Contributions by Various American Authors. In four very handsome octavo volumes of about 900 pages each, fully illustrated in black and colors. Per volume, cloth, $5; leather, $6; half Morocco, $7. *For sale by subscription only.* Full prospectus free on application.

LYMAN (HENRY M.). *THE PRACTICE OF MEDICINE.* In one very handsome octavo volume of 925 pages with 170 engravings. Cloth, $4.75; leather, $5.75.

MACKENZIE (JOHN NOLAND). *THE DISEASES OF THE NOSE AND THROAT.* Octavo, of about 600 pages, richly illustrated. *Preparing.*

MAISCH (JOHN M.). *A MANUAL OF ORGANIC MATERIA MEDICA.* Seventh edition, thoroughly revised by H. C. C. MAISCH, Ph.G., Ph.D. In one 12mo. of 512 pages, with 285 engravings. Cloth, $2.50, *net.*

MALSBARY (GEO. E.). *A POCKET TEXT-BOOK OF THEORY AND PRACTICE OF MEDICINE.* 12mo. 405 pages, with 45 illustrations. Cloth, $1.75, *net;* flexible red leather, $2.25, *net.* *Lea's Series of Pocket Text-Books.* Page 12.

MANTON (W. P.). *AN EPITOME OF OBSTETRICS.* 12mo., 265 pages, 82 illustrations. Cloth, $1.00, *net.* *Lea's Series of Medical Epitomes.* See page 10.

MANUALS. See *Students' Quiz Series,* page 14; *Students' Manuals,* page 15; *Clinical Manuals,* page 13; *Medical Epitomes,* page 10; *Pocket Text-Books,* page 12.

MARSH (HOWARD). *DISEASES OF THE JOINTS.* In one 12mo. volume of 468 pages, with 64 engravings and a colored plate. Cloth, $2. See *Series of Clinical Manuals,* page 13.

MARTIN (EDWARD.) *SURGICAL DIAGNOSIS.* One 12mo. volume of 400 pages, richly illustrated. *Preparing.*

MARTIN (WALTON) AND ROCKWELL (W. H., JR.). *A POCKET TEXT-BOOK OF CHEMISTRY AND PHYSICS.* 12mo. 366 pages, with 137 illustrations. Cloth, $1.50, *net;* flexible leather, $2.00, *net.* *Lea's Series of Pocket Text-Books.* Page 12.

McGLANNAN (A.). *AN EPITOME OF CHEMISTRY AND PHYSICS.* 12mo, 250 pages, illustrated. Cloth, $1.00, *net.* *Lea's Series of Medical Epitomes.* See page 10.

MEDICAL NEWS POCKET FORMULARY. See page 1. $1.50, *net.*

MITCHELL (JOHN K.). *REMOTE CONSEQUENCES OF INJURIES OF NERVES AND THEIR TREATMENT.* 12mo., 239 pages, 12 illustrations. Cloth, $1.75.

MITCHELL (S. WEIR). *CLINICAL LESSONS ON NERVOUS DISEASES.* 12mo., 299 pages, with 17 engravings and 2 colored plates. Cloth, $2.50.

MORRIS (MALCOLM). *DISEASES OF THE SKIN.* Second edition. 12mo., 601 pages, with 10 chromo-lithographic plates and 26 engravings. Cloth, $3.25, *net.*

MORROW (PRINCE A.). *THE RELATIONS OF VENEREAL DISEASES WITH MARRIAGE.* Octavo, about 450 pages, with many illustrations. *Preparing.*

MULLER (J.). *PRINCIPLES OF PHYSICS AND METEOROLOGY.* In one large 8vo. volume of 623 pages, with 538 engravings. Cloth, $4.50.

MUSSER (JOHN H.). *A TREATISE ON MEDICAL DIAGNOSIS,* for Students and Physicians. Fourth edition. Octavo, 1104 pages, with 250 engravings and 49 full-page colored plates. Cloth, $6.00; leather, $7.00; half morocco, $7.50, *net.*

NATIONAL DISPENSATORY. See *Stillé, Maisch & Caspari,* page 14.

NATIONAL FORMULARY. See *National Dispensatory,* page 14.

NATIONAL MEDICAL DICTIONARY. See *Billings,* page 3.

NETTLESHIP (E.). *DISEASES OF THE EYE.* Sixth American from sixth English edition. Thoroughly revised. 12mo., 562 pages, with 192 engravings, 5 colored plates, test-types, formulæ and color-blindness test. Cloth, $2.25, *net.*

NICHOLS (JOHN B.) AND VALE (F. P.). *A POCKET TEXT-BOOK OF HISTOLOGY AND PATHOLOGY.* 12mo. of 459 pages, with 213 illustrations. Cloth, $1.75, *net;* flexible leather, $2.25, *net.* *Lea's Series of Pocket Text-Books.* Page 12.

NORRIS (WM. F.) AND OLIVER (CHAS. A.). *TEXT-BOOK OF OPHTHALMOLOGY.* In one octavo volume of 641 pages, with 357 engravings and 5 colored plates. Cloth, $5; leather, $6.

OWEN (EDMUND). *SURGICAL DISEASES OF CHILDREN.* In one 12mo. volume of 525 pages, with 85 engravings and 4 colored plates. Cloth, $2. See *Series of Clinical Manuals,* page 13.

PARK (WILLIAM H.). *BACTERIOLOGY IN MEDICINE AND SURGERY.* 12mo., 688 pages, 87 engravings in black and colors, 2 colored plates. Cloth, $3.00, *net.*

PARK (ROSWELL), Editor. *A TREATISE ON SURGERY,* by American Authors. For Students and Practitioners of Surgery and Medicine. Third edition. In one large octavo volume of 1408 pages, with 692 engravings and 64 plates. Cloth, $7.00; leather, $8.00, *net.* Published also in 2 volumes at $3.75, *net,* per volume, cloth.

PARVIN (THEOPHILUS). *THE SCIENCE AND ART OF OBSTETRICS.* Third edition. In one handsome octavo volume of 677 pages, with 267 engravings and 2 colored plates. Cloth, $4.25; leather, $5.25.

PEPPER'S *SYSTEM OF MEDICINE.* See page 2.

PEPPER (A. J.). *SURGICAL PATHOLOGY.* In one 12mo volume of 511 pages, with 81 engravings. Cloth, $2. See *Students' Series of Manuals*, page 15.

PICK (T. PICKERING). *FRACTURES AND DISLOCATIONS.* In one 12mo. volume of 530 pages, with 93 engravings. Cloth, $2. See *Series of Clinical Manuals*, p. 13.

PLAYFAIR (W. S.). *THE SCIENCE AND PRACTICE OF MIDWIFERY.* Seventh American from the Ninth English edition. Octavo, 700 pages, with 207 engravings and 7 full page plates. Cloth, $3.75; leather, $4.75, *net.*

POLITZER (ADAM). *A TEXT-BOOK OF THE DISEASES OF THE EAR AND ADJACENT ORGANS.* Third American from the Fourth German edition. In one octavo volume of 896 pages, with 346 engravings. Cloth, $7.50, *net. Just Ready.*

POCKET FORMULARY. Fifth edition. See page 1.

POCKET TEXT-BOOK SERIES covers the entire domain of medicine in eighteen volumes of 350 to 525 pages each, written by teachers in leading American medical colleges. Issued under the editorial supervision of Bern B. Gallaudet, M.D., of the College of Physicians and Surgeons, New York. Thoroughly modern and authoritative, concise and clear, amply illustrated with engravings and plates, handsomely printed and bound. The series is constituted as follows: Anatomy; Physiology; Chemistry and Physics; Histology and Pathology; Materia Medica, Therapeutics, Medical Pharmacy, Prescription Writing and Medical Latin; Practice; Diagnosis; Nervous and Mental Diseases; Surgery; Venereal Diseases; Skin Diseases; Eye, Ear, Nose and Throat; Obstetrics; Gynecology; Diseases of Children; Bacteriology; Nursing; Massage. Special circular free on application.

POTTS (CHAS. S.). *A POCKET TEXT-BOOK OF NERVOUS AND MENTAL DISEASES.* 12mo. of 455 pages, with 88 illustrations. Cloth, $1.75, *net;* flexible leather, $2.25, *net.* Lea's Series of Pocket Text-Books, page 12.

—— *A TEXT-BOOK ON MEDICINE AND SURGICAL ELECTRICITY.* Octavo, about 350 pages, amply illustrated. *Shortly.*

POSEY (W. C.) AND WRIGHT (JONATHAN). *A TREATISE ON THE EYE, NOSE, THROAT AND EAR.* Octavo, 1251 pages, richly illustrated with 650 engravings and 35 plates in black and colors. Cloth, $7.00; leather, $8.00, *net. Just Ready.* Published also in two volumes. Vol. I., Posey on the Eye. Cloth, $4.00, *net.* Vol. II., Wright on the Nose, Throat and Ear. Cloth, $3.50, *net.*

PROGRESSIVE MEDICINE. See page 1. Per annum, $10.00.

PURDY (CHARLES W.). *BRIGHT'S DISEASE AND ALLIED AFFECTIONS OF THE KIDNEY.* In one octavo volume of 288 pages, with 18 engravings. Cloth, $2.

PYE-SMITH (PHILIP H.). *DISEASES OF THE SKIN.* In one 12mo. volume of 407 pages, with 28 illustrations, 18 of which are colored. Cloth, $2.

QUIZ SERIES. See *Students' Quiz Series*, page 14.

RALFE (CHARLES H.). *CLINICAL CHEMISTRY.* In one 12mo. volume of 314 pages, with 16 engravings. Cloth, $1.50. See *Students' Series of Manuals*, page 14.

REMSEN (IRA). *THE PRINCIPLES OF THEORETICAL CHEMISTRY.* Fifth edition thoroughly revised. In one 12mo. volume of 326 pages. Cloth, $2.

REYNOLDS (EDWARD) AND NEWELL (F. S.). *MANUAL OF PRACTICAL OBSTETRICS.* Octavo, 531 pages, 253 engravings and 3 plates. Cloth, $3.75, *net. Just Ready.*

RICHARDSON (MAURICE H.). *A PRACTICAL TREATISE ON ABDOMINAL SURGERY.* Octavo, about 800 pages, profusely illustrated with engravings and colored plates. *Preparing.*

RICHARDSON (BENJAMIN WARD). *PREVENTIVE MEDICINE* In one octavo volume of 729 pages. Cloth, $4.

ROBERTS (JOHN B.). *THE PRINCIPLES AND PRACTICE OF MODERN SURGERY.* Second edition. In one octavo volume of 838 pages, with 474 engravings and 8 plates. Cloth, $4.25, *net;* leather, $5.25, *net.*

ROCKWELL (W. H., Jr.). *A POCKET TEXT-BOOK OF ANATOMY.* 12mo., 600 pages, illustrated. Cloth, $2.25; flexible leather, $2.75, *net.* *Lea's Series of Pocket Text-Books.* Page 12.

ROSS (JAMES). *THE DISEASES OF THE NERVOUS SYSTEM.* Octavo, 726 pages, with 184 engravings. Cloth, $4.50; leather, $5.50.

SCHAFER (EDWARD A.). *THE ESSENTIALS OF HISTOLOGY, DESCRIPTIVE AND PRACTICAL.* Sixth edition. Octavo, 426 pages, with 463 illustrations. Cloth, $3, *net.*

—— *A COURSE OF PRACTICAL HISTOLOGY.* Second edition. In one 12mo. volume of 307 pages, with 59 engravings. Cloth, $2.25.

SCHALEK (ALFRED). *AN EPITOME OF DERMATOLOGY.* 12mo., 225 pages, 34 illustrations. Cloth, $1.00, *net.* *Lea's Series of Medical Epitomes.* See page 10.

SCHLEIF (WM.). *A POCKET TEXT-BOOK OF MATERIA MEDICA, THERAPEUTICS, PRESCRIPTION WRITING, MEDICAL LATIN AND MEDICAL PHARMACY.* Second edition. 12mo., 380 pages. Cloth, $1.75; flexible leather, $2.25, *net.* *Lea's Series of Pocket Text-Books.* Page 12.

SCHMAUS (HANS.) AND EWING (JAMES). *PATHOLOGY AND PATHOLOGICAL ANATOMY.* Sixth edition. Octavo, 602 pages, with 351 illustrations, including 34 colored inset plates. Cloth, $4.00, *net.* *Just Ready.*

SCHMIDT (LOUIS E.). *AN EPITOME OF GENITO-URINARY AND VENEREAL DISEASES.* 12mo, 249 pages, 21 illustrations. Cloth, $1.00, *net.* *Lea's Series of Medical Epitomes.* See page 10.

SCHREIBER (JOSEPH). *A MANUAL OF TREATMENT BY MASSAGE AND METHODICAL MUSCLE EXERCISE.* Translated by WALTER MENDELSON, M.D., of New York. Octavo, 274 pages, with 117 engravings.

SCOTT (R. J. E.). See State Board Examinations, page 14.

SENN (NICHOLAS). *SURGICAL BACTERIOLOGY.* Second edition. In one octavo volume of 268 pages, with 13 plates, 10 of which are colored, and 9 engravings. Cloth, $2.

SERIES OF CLINICAL MANUALS. A Series of Authoritative Monographs on Important Clinical Subjects, in 12mo. volumes of about 550 pages, well illustrated. The following volumes are now ready: CARTER and FROST'S Ophthalmic Surgery, $2.25; MARSH on Diseases of the Joints, $2; OWEN on Surgical Diseases of Children, $2; PICK on Fractures and Dislocations, $2.

For separate notices, see under various authors' names.

SERIES OF POCKET TEXT-BOOKS. See page 12.

SERIES OF STATE BOARD EXAMINATIONS. See page 14.

SERIES OF STUDENTS' MANUALS. See page 15.

SIMON (CHARLES E.). *CLINICAL DIAGNOSIS, BY MICROSCOPICAL AND CHEMICAL METHODS.* Fourth revised edition. Octavo, 608 pages, with 139 engravings and 19 full-page plates in colors. Cloth, $3.75, *net.*

—— *PHYSIOLOGICAL CHEMISTRY.* In one octavo volume of 453 pages. Cloth, $3.25, *net.*

SIMON (W.). *MANUAL OF CHEMISTRY.* A Guide to Lectures and Laboratory Work. A Text-book specially adapted for Students of Medicine and Pharmacy. Seventh edition. In one 8vo. volume of 613 pages, with 64 engravings and 8 plates showing colors of 64 tests and a spectra plate. Cloth, $3.00, *net.*

SLADE (D. D.). *DIPHTHERIA; ITS NATURE AND TREATMENT.* Second edition. In one royal 12mo. volume, 158 pages. Cloth, $1.25.

SMITH (J. LEWIS). *THE DISEASES OF INFANCY AND CHILDHOOD.* Eighth edition, thoroughly revised and rewritten and greatly enlarged. 8vo., 983 pages, with 273 illustrations and 4 full-page plates. Cloth, $4.50; leather, $5.50.

SMITH (STEPHEN). *OPERATIVE SURGERY.* Second and thoroughly revised edition. In one octavo vol. of 892 pages, with 1005 engravings. Cloth, $4; leather, $5.

SOLLY (S. EDWIN). *A HANDBOOK OF MEDICAL CLIMATOLOGY* Octavo, 462 pages, with engravings and 11 full-page plates. Cloth, $4.00.

STARR (M. ALLEN). *A TREATISE ON ORGANIC NERVOUS DISEASES.* Octavo, about 700 pages, with 300 engravings and 27 colored plates. *Preparing.*

STATE BOARD EXAMINATION SERIES. Classified and Edited by R. J. E. Scott, A.M., M.D. A series of handy, well arranged, 12mo. volumes of about 200 pages each, bound in flexible cloth and printed on paper suitable for either pen or pencil, every other page being left blank for memoranda. The respective volumes cover the subjects of Anatomy; Physiology and Hygiene; Chemistry; Surgery; Obstetrics; Pathology and Diagnosis; Practice, Materia Medica and Therapeutics. *Shortly.*

STILLE (ALFRED). *CHOLERA; ITS ORIGIN, HISTORY, CAUSATION, SYMPTOMS, LESIONS, PREVENTION AND TREATMENT.* In one 12mo. volume of 163 pages, with a chart showing routes of previous epidemics. Cloth, $1.25.

——— *THERAPEUTICS AND MATERIA MEDICA.* Fourth and revised edition. In two octavo volumes, containing 1936 pages. Cloth, $10.

STILLE (ALFRED), MAISCH (JOHN M.) AND CASPARI (CHAS. JR.). *THE NATIONAL DISPENSATORY:* Containing the Natural History, Chemistry, Pharmacy, Actions and Uses of Medicines, including those recognized in the latest Pharmacopœias of the United States, Great Britian and Germany, with numerous references to the French Codex. Fifth edition, revised and enlarged in accordance with and embracing the new *U. S. Pharmacopœia*, Seventh Decennial Revision. With Supplement containing the new edition of the *National Formulary.* Imperial octavo, 2025 pages, with 320 engravings. Cloth, $7.25; leather, $8. With Thumb Index. Cloth, $7.75; leather, $8.50.

STIMSON (LEWIS A.). *A MANUAL OF OPERATIVE SURGERY.* Fourth edition. In one royal 12mo. volume of 581 pages, with 293 engravings. Cloth, $3.00, *net.*

——— *A TREATISE ON FRACTURES AND DISLOCATIONS.* Third edition. In one handsome octavo volume of 842 pages, with 336 engravings and 32 full-page plates. Cloth, $5 *net;* leather, $6, *net;* half morocco, $6.50, *net.*

STUDENTS' QUIZ SERIES. A Series of Manuals in question and answer form, for Students and Practitioners, covering the essentials of medical science. Thirteen volumes, pocket size, convenient, authoritative, well illustrated, handsomely bound in cloth, and issued at a low price. 1. Anatomy (double number); 2. Physiology; 3. Chemistry and Physics; 4. Histology, Pathology and Bacteriology; 5. Materia Medica and Therapeutics; 6. Practice of Medicine; 7. Surgery (double number); 8. Genito-Urinary and Venereal Diseases; 9. Diseases of the Skin; 10. Diseases of the Eye, Ear, Throat and Nose; 11. Obstetrics; 12. Gynecology; 13. Diseases of Children. Price, $1 each, except Nos. 1 and 7, *Anatomy and Surgery*, which being double numbers are priced at $1.75 each. Full specimen circular on application to publishers.

STUDENTS' SERIES OF MANUALS. A Series of Manuals by Eminent Teachers or Examiners. The volumes are pocket-size 12mos. of from 300–540 pages, profusely illustrated, and bound in red limp cloth. The following volumes may now be announced: HERMAN'S First Lines in Midwifery, $1.25; BRUCE'S Materia Medica and Therapeutics (sixth edition), $1.50, *net;* KLEIN'S Elements of Histology (5th edition), $2.00, *net;* PEPPER'S Surgical Pathology, $2; TREVES' Surgical Applied Anatomy, $2.00; RALFE'S Clinical Chemistry, $1.50.

For separate notices, see under various authors' names.

STURGES (OCTAVIUS). *AN INTRODUCTION TO THE STUDY OF CLINICAL MEDICINE.* In one 12mo. volume. Cloth, $1.25.

SUTER (W. NORWOOD). *A MANUAL OF REFRACTION AND MOTILITY.* 12mo., 382 pages, 101 engravings, 4 colored plates. Cloth, $2.00, *net. Just Ready.*

SUTTON (JOHN BLAND). *SURGICAL DISEASES OF THE OVARIES AND FALLOPIAN TUBES.* Including Abdominal Pregnancy. In one 12mo. volume of 513 pages, with 119 engravings and 5 colored plates. Cloth, $3.

SZYMONOWICZ (L.) AND MacCALLUM (J. BRUCE). *A TEXT-BOOK OF HISTOLOGY OF THE HUMAN BODY:* including Microscopical Technique. Octavo, 437 pages, with 277 original engravings and 57 inset plates in black and colors, containing 81 figures. Cloth, $4.75, *net. Just Ready.*

TAIT (LAWSON). *DISEASES OF WOMEN AND ABDOMINAL SURGERY.* Vol. I. contains 554 pages, 62 engravings, and 3 plates. Cloth, $3.

TAYLOR (ALFRED S.). *MEDICAL JURISPRUDENCE.* From the twelfth English edition, revised by CLARK BELL, ESQ., of the N. Y. Bar. Octavo, 831 pages, with 54 engravings and 8 full-page plates. Cloth, $4.50; leather, $5.50.

TAYLOR (ROBERT W.). *GENITO-URINARY AND VENEREAL DISEASES AND SYPHILIS.* Second edition. Octavo, 720 pages, with 135 engravings and 27 colored plates. Cloth, $5.00; leather, $6.00; half morocco, $6.50, *net.*

—— *A PRACTICAL TREATISE ON SEXUAL DISORDERS IN THE MALE AND FEMALE.* Second edition. Octavo, 434 pages, with 91 engravings and 13 plates. Cloth, $3.00, *net.*

—— *A CLINICAL ATLAS OF VENEREAL AND SKIN DISEASES.* Including Diagnosis, Prognosis and Treatment. In eight large folio parts, measuring 14 x 18 inches, and comprising 213 beautiful figures on 58 full-page chromo-lithographic plates, 85 fine engravings, and 425 pages of text. Bound in one volume, half Turkey Morocco, $28. Specimen plates by mail.

TAYLOR (SEYMOUR). *INDEX OF MEDICINE.* A Manual for the use of Senior Students and others. In one large 12mo. volume of 802 pages. Cloth, $3.75.

THOMAS (T. GAILLARD) AND MUNDE (PAUL F.). *A PRACTICAL TREATISE ON THE DISEASES OF WOMEN.* Sixth edition, thoroughly revised. Octavo 824, pages, with 347 engravings. Cloth, $5; leather, $6.

THOMPSON (W. GILMAN). *A TEXT-BOOK OF PRACTICAL MEDICINE.* For Students and Practitioners. Second edition. Octavo, 1014 pages, with 59 illustrations. Cloth, $5.00, leather, $6.00, half morocco, $6.50, *net. Just Ready.*

THOMPSON (SIR HENRY). *THE PATHOLOGY AND TREATMENT OF STRICTURE OF THE URETHRA AND URINARY FISTULÆ.* From the third English edition. Octavo, 359 pages, with 47 engravings and 3 plates. Cloth, $3.50.

TIRARD (NESTOR). *MEDICAL TREATMENT OF DISEASES AND SYMPTOMS.* Handsome octavo volume of 627 pages. Cloth, $4.00, *net.*

TREVES (FREDERICK). *OPERATIVE SURGERY.* In two 8vo. volumes containing 1550 pages, with 422 illustrations. Cloth, $9; leather, $11.

TREVES (**FREDERICK**). *A SYSTEM OF SURGERY.* In Contributions by Twenty-five English Surgeons. In two large octavo volumes, containing 2298 pages, with 950 engravings and 4 full-page plates. Per set, cloth, $16.

——— *SURGICAL APPLIED ANATOMY.* New edition. 12mo., 577 pages, 80 engravings. Cloth, $2.00, *net.* *See Students' Series of Manuals, page 15.*

TUTTLE (**GEO. M.**). *A POCKET TEXT-BOOK OF DISEASES OF CHILDREN.* 12mo. 374 pages, with 5 plates. Cloth, $1.50, *net;* flexible leather, $2.00, *net.* *Lea's Series of Pocket Text-Books.* Page 12.

VAUGHAN (**VICTOR C.**) **AND NOVY** (**FREDERICK G.**). *CELLULAR TOXINS,* or the Chemical Factors in the Causation of Disease. New (4th) edition. 12mo., 480 pages with 6 engravings. Cloth, $3, *net.*

VEASEY (**CLARENCE A.**). *A MANUAL OF OPHTHALMOLOGY.* 12mo., 410 pages, 194 engravings, 10 colored plates. Cloth, $2.00, *net.* *Just Ready.*

VISITING LIST. *THE MEDICAL NEWS VISITING LIST* for 1903. Four styles: Weekly (dated for 30 patients); Monthly (undated for 120 patients per month); Perpetual (undated for 30 patients each week); and Perpetual (undated for 60 patients each week). The 60-patient book consists of 256 pages of assorted blanks. The first three styles contain 32 pages of important data, thoroughly revised, and 160 pages of assorted blanks. Each in one volume, price, $1.25. With thumb-letter index for quick use, 25 cents extra. For special combination rates see page 1.

WATSON (**THOMAS**). *LECTURES ON THE PRINCIPLES AND PRACTICE OF PHYSIC.* Fifth edition with additions by H. Hartshorne, M.D. In two 8vo. volumns of 1840 pages, with 190 engravings. Cloth, $9.

WHARTON (**HENRY R.**). *MINOR SURGERY AND BANDAGING.* Fifth edition. 12mo., 640 pages, with 509 engravings, many of which are photographic. Cloth, $3.00, *net.*

WHITMAN (**ROYAL**). *ORTHOPEDIC SURGERY.* One octavo volume of 642 pages, with 447 illustrations, mostly original. Cloth, $5.50, *net.*

WHITLA (**WILLIAM**). *DICTIONARY OF TREATMENT.* Octavo of 917 pages. Cloth, $4.

WILLIAMS (**DAWSON**). *MEDICAL DISEASES OF INFANCY AND CHILDHOOD.* Second edition specially revised for America by F. S. Churchill, A.M., M.D. Octavo, 538 pages, 52 engravings and 2 colored plates. Cloth, $3.50, *net.*

WILSON (**ERASMUS**). *A SYSTEM OF HUMAN ANATOMY.* Revised edition, octavo, 616 pages, with 397 engravings. Cloth, $4; leather, $5.

WINCKEL *ON PATHOLOGY AND TREATMENT OF CHILDBED.* In one octavo volume of 484 pages. Cloth, $4.

WOOLSEY (**GEORGE**). *APPLIED SURGICAL ANATOMY REGIONALLY PRESENTED.* Octavo, 511 pages, with 125 original illustrations in black and colors. Cloth, $5.00; leather, $6.00, *net.* *Just Ready.*

YEAR-BOOK OF TREATMENT FOR 1898. In contributions by 24 well-known medical writers. 12mo., 488 pages. Cloth, $1.50.

YOUNG (**JAMES K.**). *ORTHOPEDIC SURGERY.* In one 8vo volume of 475 pages, with 286 illustrations. Cloth, $4; leather, $5.

ZAPFFE (**FRED. C.**) *A POCKET TEXT-BOOK OF BACTERIOLOGY.* 12mo., 350 pages with 146 engravings and 7 colored plates. Cloth, $1.50; flexible leather, $2.00, *net.* *Just Ready.* *Lea's Series of Pocket Text-Books.* Page 12.

7